# Shedding Light on the Complex Picture of Psychosocial Factors that Promote Mental Health in Young People

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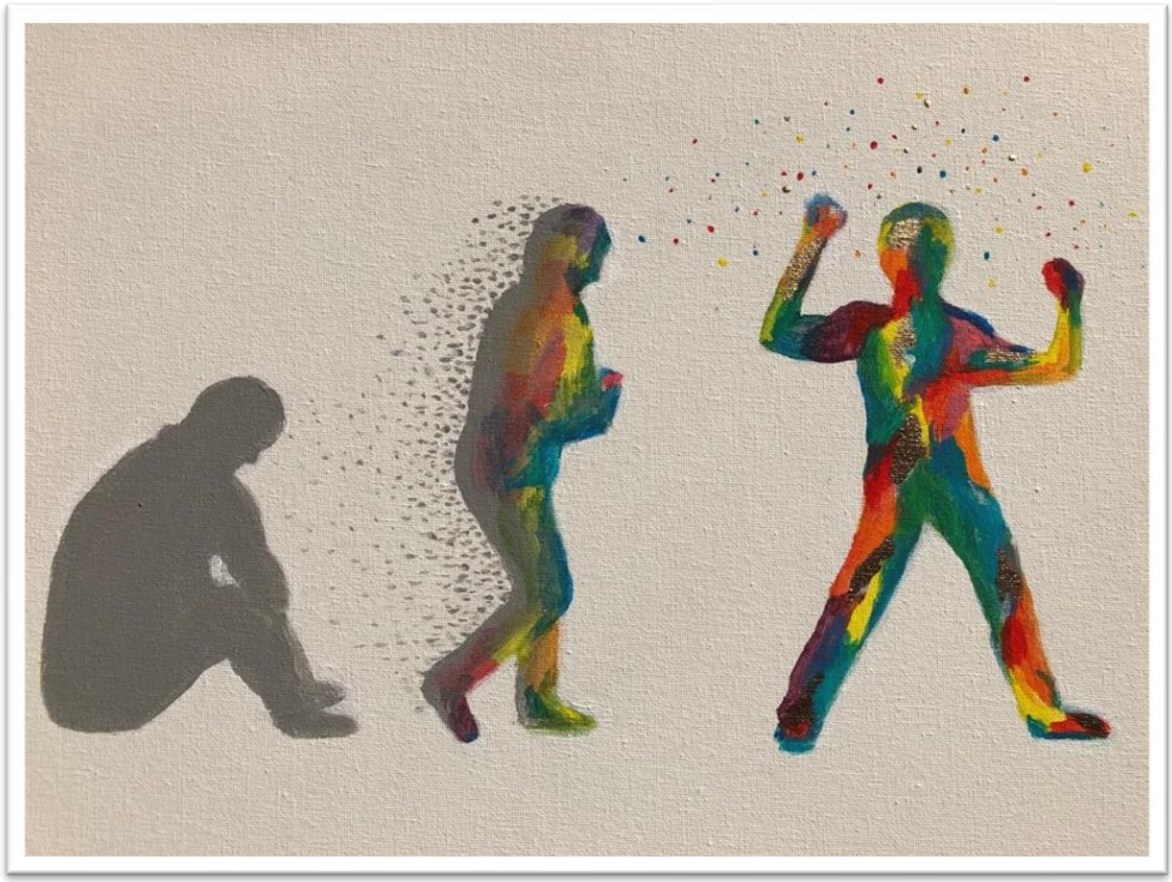
This thesis is submitted for the degree of Doctor of Philosophy.



“We work in a world of traumas and triumphs. Most of the persons we serve come to us out of necessity, struggling with the sequelae of disease and illness or the aftermath of natural or manmade disasters. We bring our expertise and compassion; they bring their bodies, minds, and compromised lives. Our worlds converge around a shared task: identifying and enhancing their capacities for daily living. ... Many intervening variables affect patients' major life changes on the one hand and illness outcome on the other. The good news is that those who rise above adversity do not belong to an exclusive club. It is not a closed system. However, some people are their own best facilitators, while others need help. … The variability of resilience may come as bad news for some, because it does not permit a simple recipe for treatment. Instead, we must commit ourselves to understanding the complexities of personality, coping capacities, and environmental influences and use them to identify goals, interventions, and environments that are meaningful ... .”

Susan B. Fine, 1991, pp. 493 – 501
https://doi.org/10.5014/ajot.45.6.493

# ACKNOWLEDGEMENTS

“Remember to look up at the stars and not down at your feet. Try to make sense of what you see and wonder about what makes the universe exist. Be curious. And however difficult life may seem, there is always something you can do and succeed at. It matters that you don't just give up.”
– Stephen Hawking –

Now that my PhD is nearing its end and all the knowledge I want to convey and share is written down, I would like to thank all those people who contributed to my work, and those who supported and nurtured me throughout these unique years.

They say doing a PhD is like a rollercoaster ride, plenty of rapidly changing highs and lows. This captures my own experience quite well: A range of marvellous days where you cannot quite believe you live in such a magical and unique place as Cambridge, to study exactly those research questions you had envisioned and carefully evolved. Days on which you meet inspiring people, or on which you find yourself dining at a long, old table, in an unrealistically beautiful and mysterious college hall. I think this is what they call ‘the Cambridge Experience’. Days on which you are met with respect and appreciation, or are given an exceptional opportunity. There are days on which you learn so many new skills which nourish your self-confidence and self-reliance. There are days on which people who you admire cite your work, and you cannot really believe why someone that brilliant would use your work to substantiate their own work. It is exciting and turbulent.

Then there are days when you sit behind your computer screen, long hours, and then longer hours, losing track of time, little interactions, unhealthy food, far too much coffee, and still more work to do. Days with plenty of problems to solve. Days on which you wonder whether the to-be-solved problems are challenges to be overcome or obstacles to give up on. There are days on which others overlook your efforts. Days on which you fear you lose self-confidence. There are days on which you wonder whether your work can actually contribute anything to society after all. Those days are daunting and exhausting.

And then there are days on which you have both: the delight and the sorrow – the rapid and the slow. Even though I may not have remembered on every single day that doing a PhD is a privilege, I surely did know deep down, and would like to wholeheartedly thank everyone who has put me into this position or has accompanied me on my PhD journey.

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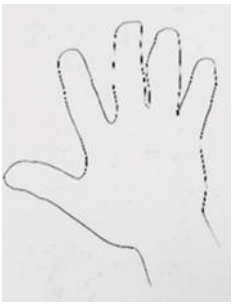

Many thanks to those people who have contributed to the behind-the-scenes work. First of all, thank you to all participants of the ROOTS and the RESIST study, and to the ROOTS study team. A big shout out to all the marvellous methods developers without whom I could not have conducted my analyses. Thank you for sharing your work openly and for enabling us applied scientists to do cutting-edge research. Moreover, thank you peer-reviewers, for all your critical, but pleasant, and truly helpful feedback. The effort you have put in is so much appreciated and has surely helped me to become a better scientist. Also, thanks to everyone who has provided helpful ideas and feedback during conferences and training courses. Thank you, Open Science Team, for the many discussions on open science best practice and publication licenses. You have been a great help and reassurance. Last but not least, thank you Gisela Kaddatz and Corinna Schnabel for creating such an utterly beautiful painting for the preface of my thesis.

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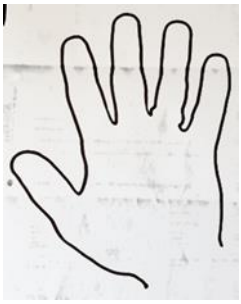

There are so many things I would like to say, so many things that have contributed to who I am and where I am standing now, but somehow it always comes down to ‘thank you for raising me up, for enabling me to struggle and to flourish’.

Thank you to everyone who has enabled me to pursue my dreams.

“Sophie: Dreams are so quick!
BFG: Yeah, on the outside. They're long on the inside.”
– Walt Disney movie based on Roald Dahl’s the BFG –

# DECLARATION

I hereby declare that my thesis entitled “Shedding Light on the Complex Picture of Psychosocial Factors that Promote Mental Health in Young People” is the result of my own work and includes nothing which is the outcome of work done in collaboration except as declared below and specified in the text. It is not substantially the same as any work that I have submitted, or, is being concurrently submitted for a degree, diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared here and specified in the text. I further state that no substantial part of my thesis has already been submitted, or, is being concurrently submitted for any such degree, diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared here and specified in the text. It does not exceed the prescribed word limit of 71000 words (excluding the front page, the preface, the acknowledgements, the declaration, the thesis abstract, the table of contents, all figures/ figure captions, all tables/ table notes, the bibliographies, all appendices, and all online supplements).

For Chapter 1 and 8 (i.e. the introduction and discussion), I devised the scientific scope and conducted the write-up. For Chapter 2, I conceived the research idea, wrote the preregistration, led the review procedure (e.g. literature search, screening, data extraction and quality ratings, as detailed below), and conducted the write-up. For Chapters 3 to 7, I conceived the general research ideas, formulated the hypotheses, performed the analyses, and conducted the write-up. Data for Chapters 3 to 6 was already collected. For Chapter 7, I designed my own study, obtained ethical approval, set up the online data collection and led the project administration (e.g. recruitment, participant reimbursement, data anonymization and storage). Chapter 1 and 8 are predominantly written in the first-person singular. All other chapters are predominantly written in the first-person plural, given that I have published, or plan to publish, this work with the respective team of collaborators, as stipulated below. Accordingly, parts of my thesis have been submitted as preregistrations, preprints and/ or journal articles and have been published under open access agreements. Below I will per chapter, if applicable, (1) provide the link to the corresponding preregistration, preprint, and/or journal article, (2) name collaborators who have directly contributed to the chapter and acknowledge colleagues who have commented on the chapter, (3) state the data source, as well as provide links to the data and the analysis code, (4) point out previous analytic work (i.e. the computation of variables) that has been used for creating the respective chapter, and will (5) specify if chapters have additional online supplements that have not been printed into the appendix. I would also like to declare that several people contributed to my work through providing helpful advice in the form of publication peer-review or at conferences and training courses.

Chapter 1

1. this work has neither been preregistered, preprinted, or been submitted for publication
2. this work has kindly been commented on by Dr. Paul Wilkinson and Dr. Anne-Laura van Harmelen
3. this chapter does not contain data
4. there is no previous analytic work that has directly been used for creating this chapter
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Chapter 2

1. this work has been preregistered, preprinted, and has been published in *Frontiers in Psychiatry* (Special Issue: Resilience, Life Events, Trajectories and The Brain, 9: 230):
    - preregistration: https://www.crd.york.ac.uk/PROSPERO/display_record.asp?ID=CRD42016051978
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2. this work is co-authored by Anne de Graaff, Helen Caisley, Dr. Anne-Laura van Harmelen and Dr. Paul Wilkinson
    - contributions to the reviewing procedure: Anne, Helen and I conducted independently the first literature screening (N = 1969); Anne and I conducted independently the literature re-screening (N = 220); Anne and I conducted independently the data extraction and quality ratings; Paul and I conducted independently the post hoc quality ratings for statistical analyses; any disagreement or uncertainty was discussed, if necessary including Paul
3. this chapter does not contain data (except for data extracted from the reviewed literature)
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Chapter 3

1. this work has been preprinted, and has been published in *Scientific Reports* (8: 15774):
    - preprint: https://osf.io/hvtng/
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2. this work is co-authored by Dr. Eiko Fried, Prof. Ian Goodyer, Dr. Paul Wilkinson, and Dr. Anne-Laura van Harmelen
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4. there are two variables that were created for previous publications, which I have re-used, details can be found in the chapter itself:
    - the childhood adversity variable: https://pubmed.ncbi.nlm.nih.gov/21736727/
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    - the childhood adversity variable: https://pubmed.ncbi.nlm.nih.gov/21736727/
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    - the childhood adversity variable: https://pubmed.ncbi.nlm.nih.gov/21736727/
5. this chapter has an online supplement:
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4. there is one variable that was created for previous publications, which I have re-used, details can be found in the chapter itself:
    - the childhood adversity variable: https://pubmed.ncbi.nlm.nih.gov/21736727/
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Chapter 7

1. this work has been preregistered, is partially based on a preprint which has been submitted for publication, other parts have not been preprinted yet:
    - preregistered: https://osf.io/wxv8q/
    - (parts have been used for a) preprint: https://osf.io/mt346/
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Chapter 8

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Jessica Fritz, September 2020

# ABSTRACT

Across the world about half a billion people suffer from mental health problems each year. Most of such mental distress starts to manifest before or during adolescence. Childhood adversity (CA) is strongly associated with mental health problems. Resilience factors (RFs), such as self-esteem or social support, reduce mental health problems following CA. While a multitude of knowledge exists for single RFs, a more holistic understanding of the RF landscape is lacking. Such knowledge is however crucial, as we otherwise miss out on important interrelations between RFs (e.g. family support → self-esteem → friendships), and as a focus on single RFs may overestimate their importance and ecological validity. With my doctoral research I aimed to take on this challenge, by conducting six projects. I focused on emotional, social, behavioural, and cognitive factors, rather than on the underlying biological or higher-order cultural factors, to specifically study those RFs that can be directly targeted in psychosocial interventions.

The first project is a preregistered systematic review with which I aimed to identify empirically supported RFs, on the individual-, family- and community-level. Building on the notion that examining single RFs may not be sufficient to understand the system that protects individuals from developing mental health problems, the second project was aimed at examining the interrelations of those 10 RFs that were identified in the systematic review and were assessed in our population-based adolescent cohort (N > 1K). With the third project I took the research question of how RFs are interconnected a step further and tried to unravel RF changes during the vulnerable period between early (age 14) and later adolescence (age 17). The fourth project was aimed at shedding light on the relative importance as well as on the predictive value with which RFs reduce subsequent mental health problems, as such knowledge may inform risk and mental-health screening. In the fifth project I explored *how* RFs mitigate the relationship between CA and subsequent mental health problems, by testing a series of direct-effect, moderation and mediation models. Such information may be vital as different effects may hold different implications for intervention research. The sixth and last project was aimed at investigating the most important RFs from the previous projects, high self-esteem and low brooding, in response to a natural stressor. More specifically, I studied those two RFs before, during and after a stress-inducing exam period in medical students, to find out whether the RFs change from before to after the stressor, and whether they co-evolve with mental distress (i.e. mutual change).

My doctoral research revealed that RFs indeed cut across multiple ecological levels (i.e. individual, family and community level) and that every level has a notable impact on mental health. This clearly underpins the importance of more holistic RF research. Moreover, I showed that RFs can be described as a promotive system. The RFs seemed to enhance each other less in adolescents with a CA history, compared to adolescents without a CA history. This potential disadvantage of the RF system in adolescents with CA was only detected at age 14, more proximally after CA. However, most pathways between RFs and distress did not change from earlier to later adolescence, which indicates that some deleterious effects that are associated with CA do not seem to wane over the course of adolescence. Furthermore, I found that brooding (abstract, negative-focussed, and repetitive thinking) and self-

esteem (quest for nurturing and optimizing self-worth) seem to be particularly promising transdiagnostic factors for risk and mental-health screening. Those RFs had the highest relative importance and predicted subsequent distress similarly well as distress could predict itself. Brooding and self-esteem were also among those RFs that best described the deleterious relationship between CA and subsequent mental health, and may therefore be fruitful targets for psycho-education as well as for psychosocial intervention research. Last but not least my research revealed that both high self-esteem and low brooding before exams mitigate increase in mental distress during the stress-inducing exams, suggesting that both have a potentially promising prevention effect. Moreover, self-esteem during exams fostered recovery of mental distress after exams, suggesting that self-esteem may also be a fruitful target for treatments at times of stress. Of course, all findings need replication in independent samples, and derived conjectures need to be tested in translational (intervention) studies.

All in all, my doctoral research has not only enhanced the empirical understanding of the complex landscape of RFs, but has also shed light on potentially time-efficient and strength-based RF targets. Therefore, my findings offer valuable recommendations for public mental health and clinical intervention research.

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# CHAPTER 1

# Shedding Light on the Complex Picture of Psychosocial Factors that Promote Mental Health in Young People

Across the world about one in four people suffer from mental health problems and according to the World Health Organization (WHO) “the magnitude of mental health burden is not matched by the size and effectiveness of the response it demands” (p. 1, report of the World Health Organization, 2001). Along those lines, I ambitiously started my doctoral research with the aim to understand how we can better protect people from succumbing to mental health problems. The deeper I dove into the topic, the more I was uncertain whether I would better fit into Konrad Lorenz’s definition of scientists, “people who know more and more about less and less”, or his definition of philosophers “people who know less and less about more and more” (quoting Konrad Lorenz, as cited on p. 1 in Logan, 2018). Nonetheless, determined to arrive at some answers I kept studying those questions of my doctoral proposal that seemed most meaningful to me. In my thesis, I will present several robust and reliable findings I arrived at, which together – I hope – will shed some light on how we can better promote mental health. For my research projects I aimed for both a population-based frame, covering a population representative set of young people, as well as for a group-specific frame, focussing specifically on those young people with prior exposure to childhood adversity. In a nutshell, my doctoral research was aimed at identifying factors that help prevent or reduce the risk of developing mental health problems in people with and without a history of childhood adversity. Throughout the thesis I will refer to those factors as resilience-promoting factors. In the next paragraphs I will explain the concepts of “mental health problems”, “childhood adversity”, and “resilience”, and will try to shed some light on the ideas behind the psychological jargon. While the exact definitions of the concepts may stay somewhat obscure, the ideas behind them will hopefully make sense and be easy to grasp.

## 1.1 CONCEPTUALIZING RESILIENCE

First, I want to introduce the concept of “*resilience*”. The word resilience comes from the Latin word “resilire” which means to recoil or jump back (Kunzler, Gilan, Kalisch, Tüscher, & Lieb, 2018). Unsurprisingly, the concept of resilience is highly multidisciplinary. For example, in financial market research the term resilience has been used to describe the ability of a financial system to bounce back from a financial crisis (Battiston et al., 2016). Similarly, in ecology, the term resilience has for example been used to describe the ability of an ecosystem to recover from an undesirable state (Scheffer et al., 2015). In clinical psychology and psychiatry, the goal of resilience research is to learn “from those who do well despite adversity about how best to help those who struggle” (p. 49 in Infurna & Luthar, 2018). Thereby resilience research reverses the focus from the aetiology of psychopathology towards understanding what promotes psychological health, well-being and thriving.

# CHAPTER 1

While the aim of resilience research seems fairly clear, the exact definition has turned out to be a complicated challenge. Several psychologists and psychiatrists have defined resilience as an ability, such "as the ability to bend but not break, bounce back, and perhaps even grow in the face of adverse life experiences" (p. 2 in Southwick, Bonanno, Masten, Panter-Brick, & Yehuda, 2014), or a capacity, such as the "capacity . . . to maintain relatively stable, healthy levels of psychological and physical functioning . . . after exposure to a [potentially traumatic event]" (p. 515 in Bonanno, Westphal, & Mancini, 2011). Others have defined resilience along the lines of its process-nature, such as the "process in which [promotive and protective factors and processes] . . . work together to support individuals to regain, sustain, or improve their mental wellbeing in contexts of adversity" (p. 2 in Ungar & Theron, 2020). And some psychologists and psychiatrists have established definitions that put the focus primarily on the multifarious nature of resilience (e.g. "resilience is defined by the context, the population, the risk, the promotive factor, and the outcome" p. 404 in Fergus & Zimmerman, 2005). To some degree all those definitions tap into somewhat different areas of the multifarious resilience concept, but equally they also all overlap. For example, all of the definitions cover some information with regard to the adversity component of resilience: "in the face of adverse life experiences", "after exposure to a [potentially traumatic event]", "in contexts of adversity" and "risk" (p. 515 in Bonanno et al., 2011; p. 404 in Fergus & Zimmerman, 2005; p. 2 in Southwick et al., 2014; p. 2 Ungar & Theron, 2020). Similarly, most definitions contain some form of psychological outcome: "healthy levels of psychological and physical functioning" or "mental wellbeing" (p. 515 in Bonanno et al., 2011; p. 2 in Ungar & Theron, 2020). Moreover, most definitions describe some underlying process nature of resilience: "bounce back, and perhaps even grow", "maintain relatively stable", or "regain, sustain, or improve" (p. 515 in Bonanno et al., 2011; p. 2 in Southwick et al., 2014; p. 2 in Ungar & Theron, 2020). Generally one could say that following a large amount of scientific publications and plenty of vibrant discussions on how to accurately define resilience, many researchers and clinicians have agreed that it should be defined as the process of adapting or recovering well after exposure to adversity (Kunzler et al., 2018). Clearly, however, this is only the first step on a long road, and psychological resilience is still a concept in search for a complete definition.

Unfortunately, the lack of a clear and practical definition has led to serious problems in the resilience literature, such as a remarkable heterogeneity in the operationalization and design of resilience studies (Kalisch et al., 2017), which inevitably has contributed to the replication crisis and may have hampered the progression of the field. To take on the challenge and tackle this problem, Kalisch and colleagues (2017) have established an international "resilience alliance" and have put forward a more precise definition. According to Kalisch and colleagues (2017; p. 3) resilience studies should generally (a) acknowledge the process nature of resilience following significant stress, (b) be aware that resilience therefore cannot be defined as "a trait or stable personality profile, . . . [nor as] a specific genotype or some hardwired feature of brain architecture", and should (c) specify and take into account both the adversity experience and the outcome of interest (e.g. "a good mental health outcome following an adverse life event or a period of difficult life circumstances").

Although this general definition provides a clearer theoretical framework and helps to align research, two questions remain that need to be answered by every psychological or psychiatric study

operating from a resilience framework: (a) adapting or recovering *from* what – or how do we define adversity, and (b) adapting or recovering *to* what – or how do we quantify whether someone is psychologically well. The latter part is often defined as a low level of or a low increase in mental health problems following adversity, also known as "mental health resilience" (Bonanno et al., 2011; Kalisch et al., 2017; Ungar & Theron, 2020). Child and adolescent psychologists and psychiatrists sometimes extend this definition by also quantifying behavioural and academic performance (Infurna & Luthar, 2018). Although this is very sensible, this thesis will exclusively focus on mental health problems, to keep the definitions as precise as possible. Thus, to neatly conceptualize "*mental health resilience*", I will next introduce the concepts of "*adversity*" and "*mental health problems*". Regarding adversity, I will specifically focus on exposure during the childhood and adolescent years – also known as early adverse experiences or childhood adversity, in short CA.

## 1.2 CONCEPTUALIZING CHILDHOOD ADVERSITY

Often, exposure to CA is thought of as something rare (Bonanno et al., 2011). In reality, however, about one in two people are exposed to at least one form of CA (Greif Green et al., 2010; Kessler, Davis, & Kendler, 1997; Kessler et al., 2010; McLaughlin et al., 2012). Generally, exposure to adversity can either be the result of a single traumatic event (e.g. death of a significant other), or the result of several severely stressful events (e.g. parental mental illness with significant impact on the family life). Importantly, adverse experiences can be active, called "commission" and indicates that something adverse has been added to the child's life (e.g. sexual abuse), or passive, called "omission" and indicates that something lacks from the child's upbringing resulting in adverse experiences (e.g. not providing the child with sufficient nutrition or medical care).

Historically, the CA concept is closely related to the concepts of childhood maltreatment, trauma and adverse childhood experiences (ACEs). Childhood maltreatment generally captures four forms of "commission", namely physical abuse, sexual abuse, emotional abuse, and witnessing intimate-partner violence, as well as two forms of "omission", namely emotional neglect and physical neglect (Gilbert et al., 2009). A related concept is trauma, which has been defined as "exposure to actual or threatened death, serious injury, or sexual violence" by the Diagnostic and Statistical Manuel of Mental Disorders (5th edition; DSM-5; p. 271; American Psychiatric Association, 2013). Trauma can be directly experienced, directly witnessed, or indirectly witnessed either through being informed about a traumatic experience that a significant other has been exposed to or through facilitating the processing of the traumatic event (e.g. first responders; DSM-5; American Psychiatric Association, 2013). Both the concepts of maltreatment and trauma have rather concise definitions, which on the one hand may aid clarification and assessment, but on the other hand may miss out on important other adverse experiences that can have similar bio-psycho-social consequences. Felitti and colleagues (1998) introduced a somewhat broader concept of childhood adversity in their prominent ACE study. The original ACEs concept captured seven adversities (i.e. three forms of maltreatment: physical, emotional, and sexual abuse; as well as four forms of household dysfunction: a mother being physically mistreated,

a family member with a severe mental illness, with time spent in prison, or with a substance abuse problem; Felitti et al., 1998). Later, the ACE concept was extended and additionally captured physical neglect, emotional neglect and parental separation (Centers for Disease Control and Prevention, 2019). Along those lines, several more recent studies have further extended the ACEs list, in the attempt to find a more holistic definition of CA. For example, Kessler and colleagues (2010) conducted a WHO World Mental Health Survey, covering 21 countries and more than 50,000 people, for which a definition of adversity was put forward that extended the ACEs with parental death, criminality within the family, life-threatening physical illness and economic adversity (that said, emotional abuse and neglect were not captured directly). Moreover, mistreatment of the mother was replaced with violence within the family (Kessler et al., 2010). Since then, many studies have used similar CA definitions.

Hence, the list of potentially relevant CAs has clearly been evaluated and complemented over the last decades. An extensive CA list seems like a good start, but how far will this get us? Realistically, a CA list – however extensive – is likely to leave the CA concept imprecise and perhaps even unavailing. For example, consider the proposed adversity parental divorce or separation. While for some young people the divorce or separation of parents is indeed a very stressful and adverse experience, for other young people parental separation may come with less discord within the family and rather reduces household stress and negative feelings. In other words, a definition of CA that exclusively relies on the presence versus absence of potential adversities will likely always be imperfect, as even the same form of adversity can be experienced highly differently across individuals. Therefore, some researchers have made it their goal to extend the CA definition and make it somewhat more sensitive and precise. As introduced earlier, CA can generally be defined as a single traumatic event or as several severely stressful events. McLaughlin (2016) has suggested to extend this definition by specifying that CA either comprises a notable divergence of a reasonable living situation, or a notable alteration of the anticipated living situation, and thus demands active adjustment by the child or adolescent (McLaughlin, 2016). Thereby, the CA definition contains a level of clinical significance. Based on this definition, CA is likely to impact social, emotional, behavioural and cognitive functioning and development (McLaughlin, 2016).

Besides content and conceptual consideration, there is no clear agreement with regard to how to compile CAs into one index. Some researchers compile CAs into a binary index (any versus no CA), others into an additive or cumulative index (e.g. a CA count; i.e. every form of CA counts to the same extent), and again others use a dimensional index (e.g. a factor analytic score derived from a latent adversity continuum; i.e. different forms of CA can contribute to a different extent). Generally, the justification for treating CA as an aggregated index, rather than as separate forms of traumatic events or severe stress, stems from a solid body of research showing that CAs often co-occur and that most forms are strongly associated with subsequent mental health problems (Greif Green et al., 2010; Kessler et al., 2010; McLaughlin, 2016; McLaughlin et al., 2012). This finding has been shown to generalize across countries, even across high versus low income countries (Kessler et al., 2010). In their attempt to strengthen this notion, Kendler and colleagues (1999) tried to disentangle whether the relationship between CA and affective symptoms is causal, and found that up to two-thirds of this relationship is indeed causal. Moreover, the relationship between CA and mental health problems is highly transdiagnostic and has repeatedly been shown to be similar for anxiety, depression and

substance use related disorders. Some researchers refer to this phenomenon as “latent liability for psychopathology” (McLaughlin, 2016). This liability seems to be particularly strong in the short-term after CA, but seems to persist over the lifecourse (Greif Green et al., 2010). CA is however not only associated with the liability for mental health problems, but has also been found to increase the risk for recurrent mental health problems, more complex presentations of mental health problems, and for treatment-resistance (Danese, 2019). Although not all young people who have been exposed to CA go on to develop mental health problems, research is vastly in agreement that CA – on average – has a detrimental effect on subsequent mental health.

Despite the fact that the definition of CA has been notably improved over the last decade, there are still a variety of caveats that researchers either disagree about or simply do not know enough about yet. First, some types of adversity are sometimes treated as risk factors in their own right, such as socio-economic status (SES), and are not counted towards an adversity index (Steptoe, Marteau, Fonagy, & Abel, 2019). Some researchers argue that SES increases the risk for some forms of CA, such as neglect, but does not necessarily need to be adverse itself. Others disagree and count it as adversity. Second, some researchers argue that the CA list is not complete and that for example social adversity, such as neighbourhood violence, would need to be added to an adversity index (McEwen & Gregerson, 2019). Third, the timeline for assessing CA varies heavily between studies, and there is little consensus on up to which age adversity should classify as “childhood” adversity (Schlechter, Fritz, & Wilkinson, 2019). Fourth, there is still very little knowledge as to whether it is sufficient to assess the presence versus the absence of adversity, or whether the adversity severity, frequency, chronicity and age of onset should (additionally) be taken into account. Preliminary research showed that not only the presence vs absence of CA increases the risk of mental health problems, but that the risk of developing mental health problems seems to increase as a function of the frequency and severity of the exposure (Schlechter et al., 2019). Besides the question regarding the most insightful facet of CA, research also widely differs in how to compile knowledge on CAs. As described above, CA can for example be compiled as binary, additive or cumulative, or as dimensional index. Moreover, some researchers have suggested to conceptualize nominal CA classes, and for example categorize CAs into “deprivation” and “threat” (McLaughlin, 2016). Fifth, CA is often assessed via retrospective report (e.g. asking adults whether they have been exposed to any type of adversity when growing up), and sometimes prospectively (e.g. tracking a sample of children and their caregivers while the child is growing up, and assessing CA repeatedly, such as every 2 or 3 years). Recent research found however that prospective versus retrospective CA reports only overlap very little (e.g. for maltreatment: 44 to 48%) and should therefore not be treated as interchangeable (Danese, 2019). Among many others, a potential explanation for this discrepancy is recall bias, which is likely to be lower in prospective study designs (Gilbert et al., 2009). Sixth, both retrospective and prospective reports can be measured in different ways. For example, some studies have used questionnaires, others have used (semi-structured) interviews, and again others have solely relied on official records (e.g. medical, court or social services records). Seventh, and related to the previous point, some studies assess CA via self-report by the exposed person (i.e. subjective report), while other studies use parent or teacher reports, or even official records (i.e. more objective reports). Importantly, cutting-edge research suggests that subjective, self-

reported CA is a much stronger predictor for subsequent mental health problems than objectively reported CA (Danese & Widom, 2020).

In sum, researchers need to be very careful and clear in how they define and assess the CA concept and need to be aware of the caveats that come with their decisions. In most of my empirical Chapters (Chapters 3 to 6), CA was assessed up to the age of 14 and was measured with a semi-structured interview conducted with the primary caregiver. CA was then clustered into a binary "any versus no CA" index (see Chapters 3 to 6 for more details and reasoning behind those decisions). In the general discussion in Chapter 8, I will reflect on the multifaceted and complex concept of CA, will discuss my findings in the light of their limitations and will make recommendations for future research. Although the CA concept is very complex, McLaughlin refers to it as "you know it when you see it" phenomenon (p. 363 in McLaughlin, 2016). In other words, although the concept needs some thoughtful definition, once agreed upon a definition, the assessment can be straightforward. Thus, the CA concept is not only (a) theoretically pertinent for the conceptual definition of resilience-promoting factors and (b) clinically highly relevant as individuals with exposure to CA have an increased risk for developing mental health problems, but it is also (c) a very practical concept as it often can be assessed rather straightforwardly within a clinical interview or with a self-report questionnaire. In the realm of resilience-promoting research, the CA concept facilitates the detection of promotive factors that mitigate the detrimental relationship between adversity and mental health and are advantageous for decreasing the vulnerability to mental health problems. Moreover, the CA concept enables the comparison of promotive factors between individuals with and without CA exposure.

## 1.3 CONCEPTUALIZING MENTAL HEALTH PROBLEMS

For a complete conceptualization of "*mental health resilience*", we now only need to properly define "*mental health problems*". Mental health problems are vastly prevalent all over the globe. According to Steel and colleagues (2014) about one in five people report to have suffered from common mental health problems (here covering depression, anxiety or substance use disorders) within the previous 12 months (155 surveys; $n$ = 665,433; from 59 countries), and about one in three over the course of life (85 surveys; $n$ = 463,998; from 39 countries). Put differentially, across the world about half a billion people suffer from mental health problems each year (Kunzler et al., 2018). Interestingly, there are only minor differences in the prevalence of those disorders among Western and non-Western, or low versus high income countries (Steel et al., 2014). Hence, while society is likely to influence mental health to some degree, vulnerability to mental health problems is likely to depend on a manifold of causes and triggers, including stressful life events, genes, hormones, and brain alterations. Moreover, different disorder clusters may differentially be impacted by those triggers or causes. For example, while autism and schizophrenia are likely to have a notable genetic component, the aetiology of post-traumatic stress disorder has a strong environmental component, as it is triggered by traumatic life-events; and eating disorder seems to have a noteworthy societal component as it is impacted by societal body image norms.

Although the DSM (Diagnostic Statistical Manual of Mental Disorders; American Psychiatric Association, 2013) and the ICD (International Statistical Classification of Diseases and Related Health Problems; World Health Organization, 2010) try to classify and describe “different” mental disorders as neatly as possible, there seems to be remarkable overlap between symptom clusters (Borsboom & Cramer, 2013) and many disorders have been found to co-occur (Kotov et al., 2017). The co-occurrence of disorders is known as comorbidity and has led many researchers to ask whether it at all is useful to cluster disorders into distinct categories, rather than trying to get a transdiagnostic index that captures vulnerability to many disorders. Furthermore, there is not only a large amount of overlap between disorders, but also a good amount of heterogeneity within disorder categories that should ideally be taken into account in a disorder or vulnerability index. In addition, not all symptoms are equally severe – e.g. for major depression, compare suicidal thoughts with indecisiveness (American Psychiatric Association, 2013; Cole et al., 2011). Moreover, the vast majority of research showed that mental health problems can better be modelled as linear continua rather than distinct categories (Caspi et al., 2014; Kotov et al., 2017). Similarly, not only do discrete mental health disorder categories lack reliability, they are also prone to miss out on those individuals who do not meet the required amount of symptoms for a mental disorder, but do experience a significant impairment of daily functioning (Kotov et al., 2017).

Yet, arguing for or against transdiagnostic versus disorder specific approaches is far out of the scope of this thesis. Besides, it seems likely that this question is wrong in itself and that both approaches may be helpful, only in different contexts. In this thesis, I will exclusively use transdiagnostic indices. More specifically, I will collapse a broad range of anxiety, depression, general affect and stress related symptoms into one broad symptom index. With this index I intend to capture the vulnerability to a wide range of mental health problems. In the remainder of the thesis I will refer to those indices as “general distress” or “mental distress” indicators. I believe that such a transdiagnostic general distress index is ideal for my purpose of identifying which factors help promote general mental health. A factor which succeeds in reducing the vulnerability to transdiagnostic mental health problems and perhaps even stabilizes or enhances good mental health, seems to be of wide-ranging purpose as it promises to be helpful for the majority.

## 1.4 CONCEPTUALIZING PSYCHOSOCIAL, RESILIENCE-PROMOTING FACTORS

Now that the concepts of mental health resilience, CA and mental health problems have been introduced, you may find yourself wondering which conjecture is more likely: “whatever does not kill us makes us stronger” (p. 1025 in Seery, Holman, & Silver, 2010) or rather “time does not heal; time conceals” (p. 131 in Felitti, 2009). Is it either, neither, or both? What makes some people more likely to be mentally healthy, even after severe CA experiences, than other people? What increases the chance of being resilient, staying mentally healthy or recovering to an acceptable level of mental health? Answers to those questions are surely high time, but somehow seem to be pending. One way to study those questions is to take a bird’s eye view, and to examine those factors that have been empirically shown to decrease the vulnerability to mental health problems during or following exposure to stress or

adversity. Those factors are also called "*resilience-promoting factors*". In the remainder I shall refer to them as RFs. While the link between CA and mental health problems has been shown to be rather conclusive, the role of RFs in the relationship between CA and mental health problems seems so far to be vague, as it lacks a systematic evaluation of empirical findings. In the mental health resilience literature several statistical models have been put forward to shed light on RFs. I shall briefly review three prominent types of those models, namely *trajectory-focussed*, *variable-focussed*, and *resilience-score* models. I shall then explain which conceptual modelling framework and content-based definition I have applied to study RFs.

For *trajectory-specific* models researchers often study a group of people, who all have been exposed to an adverse experience, and cluster them based on how their mental health levels develop in the aftermath of the adversity (for details see Bonanno et al., 2011). For example, Bonanno and colleagues (2011) report that those models often identify four trajectories: resilience (stable low or no mental health problems), recovery (a brief increase in mental health problems, followed by recovery to low or no mental health problems), delayed mental health problems (a continued increase in mental health problems), and chronic mental health problems (a high and rather stable level of mental health problems right after the adverse experience). The advantage of those models is that they shed light on the variability of mental health problems between people exposed to adversity and can help to identify trajectory-specific characteristics. A disadvantage is that identified promotive or protective factors that describe the trajectories cannot be compared back to people without exposure to adversity, as those are not included in these models.

For *variable-specific* models researchers study a group of people, both those who have been and who have not been exposed to adversity, and relate the adversity (either a binary score, or the adversity level) to the level of subsequent mental health problems (see Fergus & Zimmerman, 2005; or Masten, 2001). The major benefit of those models is that they can elegantly shed light on variables that can explain the relationship between adversity exposure and subsequent mental health and thereby identify which and how RFs mitigate mental health problems. As those models can shed light on how RFs operate, in both people with and without adversity exposure, they are particularly informative for translational research with a focus on prevention or treatment (Masten, 2001). It is however disadvantageous that those models do not directly take into account different mental health trajectories following adversity.

*Resilience-score* models can be conducted on both an individual- as well as on the group-level. An individual-level resilience-score model has for example been put forward by Kalisch, Müller, and Tüscher (2015). Here, one first calculates the change in mental health problems from before to after an adverse experience and then sets the change in mental health problems off against the level of adversity. This way every person gets an individual resilience-score (see Kalisch et al., 2015). A group-level resilience-score model has for example been described by van Harmelen and colleagues (2017). Here, only one assessment is needed for mental health problems, but a group of people is required. Mental health problems are set off against the level of the adverse experiences, which together provides a group-level parameter for the expected level of mental health problems given the adversity level. Every person then gets a resilience-score based on how far the person's mental health level lies above

or below the group level, given the person's adversity level (van Harmelen et al., 2017). In a next step one could then identify which RFs are associated with the resilience-scores. The advantage of those models is that every person receives one readily interpretable resilience-score. Disadvantages are that the group-level model does not take into account different mental health trajectories following adversity, and neither of the two approaches can directly model how RFs explain the relationship between adversity and mental health problems, as those components are inherent to the resilience-score and not modelled as separate entities.

Clearly, all of those models have important advantages and disadvantages, and arguably, research on mental health resilience may most proliferate if we carefully choose the right type of model for the research question at hand. That said, for some research questions the models can of course complement each other and surely do not function as polar opposites. Within my thesis I shall focus on variable-specific models, as I believe that they will enable me to find the most direct and decisive answers to my research aim of shedding light on psychosocial RFs that can promote mental health in young people with and without a history of CA. More specifically, I shall focus on two commonly applied types of variable-specific models to identify and examine RFs, namely on moderation and mediation models (Fergus & Zimmerman, 2005; Masten, 2001). In both a moderation and a mediation model the RF is set out to explain the relationship between adversity – in my thesis, CA – and subsequent mental health problems. A moderation model answers the question whether the disadvantageous effect of CA on subsequent mental health is weaker in individuals with a higher level of the RF; or to put it differently, whether the RF mitigates subsequent mental health problems more in individuals with than without CA (this is mathematically called an interaction effect). A mediation model answers the question whether the RF operates as an intermediate factor on the pathway between CA and mental health, and thereby can (at least to some degree) disrupt the disadvantageous effect of CA on subsequent mental health problems (CA → RF → mental health problems; this is mathematically called an indirect effect or indirect pathway). In both cases the idea is that a higher level of the RF mitigates the risk of subsequent mental health problems and thereby promotes mental health (Masten, 2001). As mental health resilience per definition is the adaptation to adversity, all RFs should be empirically related to exposure to adversity. Both moderation and mediation models ensure that this criterion is met, as in moderation models the RF interacts with CA and in mediation models the RF is the intermediate, indirect factor on the pathway between CA and subsequent mental health problems (Masten, 2001; Rose, Holmbeck, Millstein Coakley, & Franks, 2004). The interested reader can find a more detailed stipulation of the mathematical details of moderation and mediation effects in Chapters 2 and 6.

Importantly, defining RFs only based on this criterion would be insufficient, as it only specifies how the RFs conceptually operate and does not yet offer any content related specification. Therefore, I further set out that all RFs have to function on psychosocial domains: i.e. social, emotional, cognitive and behavioural domains. Biological factors are excluded, as the research interest of this thesis builds around the idea of conceptualizing, identifying, assessing, and better understanding RFs that can directly be targeted in psychosocial interventions. The specific RFs that will be studied throughout my thesis will be introduced in Chapter 2, which provides a systematic review with the aim to identify empirically supported, psychosocial RFs. Based on my definition, RFs can also be clustered into

individual, family and community factors that protect people from succumbing to mental health problems. This is important, as it implicates that not only intra-personal skills, but also inter-personal or external resources (i.e. those coming from friends, family and the community) can be targeted to stabilize or improve mental health. Particularly in the light of recent research indicating that external resources can influence mental health beyond intra-personal skills (McElroy et al., 2019), the inclusion of inter-personal resources seems a highly important facet for getting a more complete and ecologically valid view of RFs.

Research suggests that people, unsurprisingly, do not only possess one intra-personal skill or one inter-personal resource, but that people naturally have multiple, coalescing RF skills and resources. Therefore, studying the impact of a single RF in isolation may overestimate the effect and actual impact of the RF. More specifically, preliminary research suggests that RFs are bidirectionally influencing each other, and that their effects on mental health problems are additive or cumulative (Bonanno et al., 2011; Fergus & Zimmerman, 2005; Masten, 2001). Those findings underpin the importance of studying a range of RFs, as opposed to just a single RF, to further shed light on their potentially interactive nature and to enhance the ecological validity. Moreover, preliminary research has revealed that promotive factors may account for a similar degree of mental health problems than risk and adversity do (Patel, Flisher, Hetrick, & McGorry, 2007). This suggests that RFs may be highly powerful in remediating risk and adversity, and in stabilizing or improving mental health. Or, to put it in Zimmerman and colleagues' (2013, p. 1) words: "resilience occurs when environmental, social, and individual factors interrupt the trajectory from risk to pathology". Although the importance of RFs for promoting mental health is evident, the interactions, stability, differential importance, and mechanisms of RFs, as well as their potentially mutualistic relationship with mental health problems are not yet sufficiently understood. Such information is however crucially needed for informing translational prevention and treatment research that aims to screen, monitor, stabilize and/or increase mental health.

I believe that the provided conceptual and content definition for RFs gauges a clear and practical framework for studying RFs. Nonetheless, there remain some caveats which I want to draw some attention to. The attentive reader may wonder whether the name "*resilience*-promoting" factor is accurate, as it misleadingly may insinuate that those factors can only be promotive in individuals with a history of CA, when in fact RFs based on my definition may promote mental health in individuals *with* and *without* CA. This is an absolutely relevant question to discuss, as strictly speaking, resilience can indeed only occur in individuals exposed to adversity. I acknowledge that the name is not perfect; yet, there are two reasons which substantiate its usage. The first reason stems from the fact that all RFs that are investigated in my doctoral research are altered by CA, given that they must have an interaction (i.e. moderating RFs) and/or a direct relationship with CA (i.e. mediating RFs). Hence, the requirement that an RF must function as moderator and/or as mediator explains the term "resilience" in "resilience-promoting" factor. The second reason stems from the fact that regardless of whether the RF operates as moderator and/or as mediator, a high level of the RF should mitigate subsequent mental health problems; whether predominantly in individuals with CA, or in both individuals *with* and *without* a CA history. This reason explains the term "promoting" in "resilience-promoting" factor. Hence, together I arrived at the name "resilience-promoting" factor. The underlying idea is that enhancing the promotive

level of the RF should increase the chance of reducing (the risk for) subsequent mental health problems. An important detail, which does not justify the name “RFs” but the identification strategy, stems from the fact that precluding any type of adversity in the first place would of course be ideal, and would probably eradicate many mental health problems, but is unfortunately often unrealistic. Hence, the next best strategy is to prevent or reduce mental health problems as well as possible, which is where RFs come in (Masten, 2018).

Another important caveat is that even though I try to define RFs in such a way that we can expect that they are protective for many or perhaps even the majority of the people, no one RF will be protective for everyone. All RFs are likely to be dependent on the type(s) of adversity experienced, the person’s genetic make-up, the socio-cultural context and support system, the proneness for vulnerability to specific symptom clusters, the level of functioning (or impairment) in daily life, and potentially on the mental illness course and chronicity. Along the same vein, one may wonder whether resilience and risk factors are opposing sides of the same continuum. The most likely answer for my research is that some RFs and risk factors seem indeed to be on opposing sides of the same continuum (e.g. RF = high friendship support; risk factor = low friendship support; van Harmelen et al., 2016) whereas for others this apparent dichotomy seems more complex. For example, *high* rumination can be both an RF and a risk factor depending on whether its content is positive or negative (e.g. RF = high positive rumination & low negative rumination; risk factor = low positive rumination & high negative rumination; while high positive and high negative rumination often go together; Harding & Mezulis, 2017). The interested reader is invited to read a thorough discussion covering this topic in Appendix C.16, or a summary of this topic in my thesis discussion in Chapter 8. Yet, regardless of whether resilience and risk factors operate on the same continuum, are inversely correlated but not identical, or differ in content valence, understanding the nature of RFs seems to have universal appeal as it focuses on what promotes good mental health rather than on what increases mental health problems.

In sum, rather than zooming in into one specific RF, which would qualify as reductionism, I embrace a broad and hopefully ecologically more valid stance, through trying to understand multiple RFs and their interactions at the same time, which qualifies as complexity framework. Therefore, in contrast to using a magnifying glass and zooming in into a specific detail, as so often done in many areas of life-science research, I will take the opposite approach, namely zooming out and trying to identify overall patterns in the bigger picture. As already explicit by the metaphor, this goes at the costs of the details of single RFs, but comes with the advantage of potentially being able to understand the big picture of the psycho-social protective system better. Eventually, I hope that this approach will help us to gain a better understanding about the components that contribute to good mental health, and thus will help us to identify those components that may, from a positive psychology perspective, be promising screening, prevention and treatment targets. Instead of focussing on reducing adversity exposure or targeting mental health symptoms, the positive psychology perspective focusses on skills and resources that promote good mental health and well-being. Obviously, such an ambitious pursuit will not be met by a single research project, and also not by the six projects I present here. Yet, I believe that based on the six projects, which I each devote one chapter to, some preliminary notions can be derived that meaningfully contribute to the understanding of the complex nature of positive-based

mental health promotion. A last, but not least important, caveat I want to shed light on is that research on RFs comes with the danger that people may think that they have to be able to possess sufficient RFs to thrive (naturally), and that it is their own fault if they do not develop RF skills or recruit RF resources to be able to thrive (Infurna & Luthar, 2018). I want to make crystal-clear that the idea of this research is not to find out which RFs people naturally should possess. Instead, the idea is to find out which RFs are most advantageous and can theoretically be targeted for screening, prevention and treatment, to eventually help more people to have stable mental health or to recover to an acceptable level of mental health.

## 1.5 THE PERKS OF ADOLESCENT MENTAL HEALTH

Although knowledge on RFs is important across all lifecourse stages, the research in this thesis will focus on the adolescent and young adulthood life-periods. Patel and colleagues (2007, p. 1302) elegantly describe this population as: “Developmentally, they are emerging adults (Graham, 2004), sexually mature, in the final stages of their educational career or in the early stages of their employment career, and embarking on several socially accepted adult pursuits including finding and keeping a job, romantic relationships, and, in some cultures, using alcohol and tobacco.”. Consequently, many adolescents and young adults are faced with enduring changes and challenges they have to adapt to. Hence, it is logical that anxiety and depression related problems often first emerge during this susceptible period, with numbers as high as one in four young people succumbing to such mental disorders (Patel et al., 2007; Ries Merikangas et al., 2010). Anxiety related mental health problems often start developing during late childhood and early adolescence. Depression related mental health problems often start emerging during adolescence and young adulthood, and are found to double during the course of adolescence (Kessler et al., 2005; Ries Merikangas et al., 2010). The US National Comorbidity Survey Replication Study, in which 9,282 people were assessed, found that about 75 percent of all mental disorders start to emerge during adolescence and young adulthood (Kessler et al., 2005; Patel et al., 2007).

Hence, the most common mental health problems often develop and manifest themselves over the course of adolescence. For this reason, the adolescent and young adulthood years are known as a very vulnerable time period. Moreover, mental health problems in young people may negatively impact educational achievements, social and romantic relationships, and financial security, which all might recursively disadvantage the young people and hamper a successful start into adulthood (Patel et al., 2007). This clearly underpins the importance of supporting mental health in adolescents and young adults. Crucially, the previously delineated sensitivity does not only come with an amplified susceptibility, but also with the possibility of plasticity, and thus the chance to help young people to retain or develop good mental health. Along those lines, the adolescent and young adult period is also known for its sensitivity to family, community and environmental influences (Patel et al., 2007). Moreover, some research suggests that individuals with more exposure to CA seem to be particularly sensitive to positive family, community and environmental influences (Albott, Forbes, & Anker, 2018).

Therefore, I believe that studying intra- and inter-personal RFs in this period is highly fruitful. Yet I clearly want to point out that my research needs to be replicated in children, adults and the elderly, to shape a full picture of the relevance of RFs along the lifecourse.

## 1.6 THESIS PREVIEW

All chapters in this thesis are technically independent and can be read without knowing the other chapters. However, most chapters build on each other and follow upon each other in a logical order. Chapter 2, i.e. the one after the introduction, is a preregistered systematic review which is aimed at identifying empirically-supported RFs that reduce the risk of mental health problems in young people subsequent to CA. As introduced earlier, in the review I focus on amenable RFs that function on social, emotional, cognitive and behavioral levels, and can be targeted in therapeutic and preventative interventions. This chapter forms the basis for all other chapters as it identifies the empirically supported RFs that I will scrutinize in more depth in the remainder of the thesis. In Chapter 3, I intend to establish a network model of those RFs that were identified in Chapter 2 and were also assessed in our population-based adolescent cohort. This aim is based on the idea that examining individual RFs may not be sufficient to understand the system that protects individuals from developing mental health problems. Hence, I aim for an ecologically more valid approach, examining multiple RFs at the same time. To this end, I conceptualize the RFs as a complex system, or more specifically as a network model, to investigate whether and how the RFs interact with each other. I then ask the question whether those RF networks differ between groups of adolescents with (CA+) and without a history of CA (CA-). I expect that the RFs will be related to each other in both group networks, but that CA+ and CA- networks will be dissimilar in structure. I additionally estimate the RF network models including a general distress index, to explore the impact of general distress levels on the network structures in both the CA+ and the CA- group. In Chapter 4, I take the question of how RFs are interconnected a step further and try to unravel RF changes during the vulnerable period between early and later adolescence. More specifically, I examine whether RFs change between early (age 14) and later (age 17) adolescence, through investigating (a) RF mean levels, (b) RF interrelations, and (c) the way RFs are interrelated with distress (directly and/or indirectly via other RFs). Importantly, I explicitly examine whether RFs change differentially in the CA+ and the CA- groups.

In Chapter 5, the research question will move from understanding the nature and changes of the protective RF system to the predictive value of the RFs. Accordingly, I try to disentangle to what degree RFs can explain subsequent distress, which RFs are the best indicators for subsequent distress, and with what accuracy RFs can predict distress levels three years later. In Chapter 6, I aim to shed light on how RFs reduce subsequent mental health problems after CA. To this end, I examine whether most RFs function as a mediator, as a moderator, or as both for the positive relationship between CA and subsequent mental health problems. I will also discuss differential implications of moderator and mediator RFs for translational research. In Chapter 7, I will study those RFs that turn out to be most promising in the previous five chapters in an experimental (within-subject) cohort study, before, during

and after a natural stressor. To this end, I examine students during a normal semester phase before the exams, during their exam period, as well as after the exams (i.e. during the summer vacation for many students). More specifically, I intend to find out whether the RFs (a) change in response to the stressor, (b) co-evolve with changes in mental distress, and whether they (c) mitigate increase in mental distress during as well as foster recovery of mental distress after the stress-inducing exams. Finally, in the discussion, Chapter 8, I broadly summarize the main findings, relate them to each other, and discuss the overall limitations and implications of this thesis in depth.

## 1.7 ON THE BENEFIT OF PSYCHOSOCIAL, RESILIENCE-PROMOTING FACTORS

Now that I have explained the major aims of this thesis, I shall try to shed light on why I believe that it is fruitful to study RFs. Firstly, RFs may perhaps lend themselves well as mental-health screens. As such, they may be cost-effective and are likely to come with a low stigma risk, which could be particularly beneficial for community or school-based settings. The usefulness of RFs as mental-health screens will be explored in detail in Chapter 5 and in the general discussion, Chapter 8. Besides informing mental-health screening, RFs may also be crucial for informing psychoeducation. That is, social workers, teachers, pastoral support, counsellors, and psychotherapists may not only want to inform about symptoms (i.e. their potential origin, interpretation and treatment), but may also want to point to resources and skills that promote good mental health. Although it is very likely that day-to-day psychoeducation often includes such information already, empirical support for the actual usefulness of RFs should further underpin and potentially increase the inclusion of such information in psychoeducation. I shall discuss this topic in more depth in Chapter 8, after having discussed the findings of the separate chapters.

Moreover, knowledge on RFs is likely to be relevant for informing positive psychology interventions. Positive psychology interventions is an umbrella term for those interventions that focus on enhancing positive cognitions, emotions, and behaviours rather than decreasing symptoms (Bolier et al., 2013). Three positive psychology interventions with slightly different but prominent ideas are Seligman's Positive Psychotherapy (PPT; Seligman, Rashid, & Parks, 2006), Fava's Well-Being Therapy (WBT; Fava & Tomba, 2009), and Padesky and Mooney's Strength-Based Cognitive-Behavioural Therapy (SBCBT; Padesky & Mooney, 2012). PPT interventions generally focus on fostering positive feelings, intra-personal skills and resources, as well as a meaningful life (Seligman et al., 2006). WBT focusses on not reducing and overcoming symptoms, but fostering or reinstating well-being, aiming for a full recovery and reducing the risk of relapse (Fava & Tomba, 2009). SBCBT focusses on the patient's very own strengths instead of weaknesses (Padesky & Mooney, 2012). Whereas SBCBT is designed as person-specific intervention approach (Padesky & Mooney, 2012), WBT and PPT can be set up as group interventions, which for example can be delivered as school or community-based training (Bolier et al., 2013; Fava & Tomba, 2009). Moreover, several forms of PPT have been developed that can be delivered as self-help trainings, also via the internet. Hence, those forms of resilience promoting interventions appear to have a low stigma risk, and particularly group or

online-based positive psychology interventions seem to be not only easily accessible but also cost-effective (Bolier et al., 2013; Seligman et al., 2006). Whereas PPT and WBT have been shown to be effective as preventative interventions (Bolier et al., 2013; Fava & Tomba, 2009), SBCBT is particularly designed as treatment intervention or CBT supplement (Padesky & Mooney, 2012). All three forms (PPT, WBT and SBCBT) are suggested to be effective for relapse prevention purposes (Bolier et al., 2013; Fava & Tomba, 2009; Padesky & Mooney, 2012).

The pressing question is, however, how knowledge on RFs can inform those (and other) interventions, as this will answer how RFs may be able to inform translational intervention research. I shall try to shed light on this question in Chapter 8, taking into account the findings of the separate chapters. Generally, knowledge on RF skills and resources may help to identify the most important amenable factors that promote and/or stabilize mental health and well-being. Moreover, knowledge on differences in RFs between groups of adolescents with and without CA may be particularly informative when targeting high-risk populations, as for example interventions aimed at only those individuals who come from disadvantaged backgrounds (Zimmerman et al., 2013), as it then is crucial to know which RFs are most helpful following exposure to CA. Similarly, knowledge on RFs can also be highly informative when it cannot (ethically or practically) be assessed which individuals have a history of CA, such as in school-based group interventions. For those settings particularly those RFs that perform well for both individuals with and without exposure to CA seem interesting intervention targets. Importantly, the knowledge of RFs discussed in this thesis can only inform about which RFs may potentially be fruitful targets in (relapse) prevention and/or treatment, but translational research is crucially needed to (a) test the actual usefulness of RFs in action and to (b) determine which RFs can help best in which setting.

Last but not least, empirical knowledge on RFs may also be crucial for enhancing resilience theory. So far, there has been a vital discussion on how to define resilience, there is an ongoing discussion on how to best measure resilience, and there exists a cascade of independent research on single RFs (Kunzler et al., 2018). Importantly however, despite the fact that it has been regularly suggested that RFs are interrelated and do not function is isolation (Diehl, Hay, & Chui, 2012), little research has tried to understand the bigger picture of how a manifold of RFs may operate as a protective system. Similarly, it is often clearly pointed out that it may not be sufficient to change the individual when promoting mental health, but that a change in the individual's environment may be required as well (Diehl et al., 2012). Yet, to the best of my knowledge, there is little research on how intra-personal skills and inter-personal resources interact with each other, which however may be a crucial addition to the ecological validity of resilience knowledge and theory. Moreover, there seems so far to be little knowledge on whether RFs change from prior to during and after stress exposure. Equally, more knowledge is needed on whether RFs change in conjunction with mental distress. Studying those questions is critical for resilience theory and research as it elucidates whether and how different ecological levels (i.e. individual, family and community) interact, sheds light on the potentially fluctuating nature of RFs, and as it aids the identification of the time frame in which RFs can be most effective.

## 1.8 CONCLUDING REMARKS

All in all, the presented research aims to understand the complex nature of RFs, with the goal to provide and extend empirical knowledge on intra- and inter-personal skills and resources that promote mental health. Having discussed all the research aims and the intended implications it is time to point out the (to be) expected limitations. First and foremost, I want to point out that I intend to keep the applied methods as state-of the art and robust as possible, to improve accuracy and replicability of the findings. Yet most psychological methodology is, and will always be, imperfect. Statisticians and psychometricians often describe this with the aphorism “all models are wrong but some are useful” (p. 2 in Box, 1979). Similarly, the reasoning and interpretation that seems correct to me today, may at some point turn out to be mistaken or only half the story. By saying this I do not want to discourage the reader to follow on, I just want to point to realistic limitations upfront. In sum, I will aim to the best of my ability to shape an as accurate, robust, and reliable picture of the findings to inform epidemiological, experimental and translational future research as soundly as possible, but “possible answers suggested here are examples, not conclusions; some will turn out to be wrong” (Nesse, 2019, p. xii).

# CHAPTER 2

# A Systematic Review of Amenable Resilience Factors that Moderate and/or Mediate the Relationship between Childhood Adversity and Mental Health in Young People

Up to half of Western children and adolescents suffer from at least one type of childhood adversity (CA; Greif Green et al., 2010). As explained in Chapter 1, CAs span a wide range of traumatic and stressful experiences, and are associated with an increased risk for subsequent psychopathology (Greif Green et al., 2010; Kessler et al., 2010). Recently, a World Health Organization study, based on data from 21 countries (*N* = 51945), showed that approximately 30% of all mental health problems are attributable to CA (Kessler et al., 2010). Fortunately, not all individuals who have experienced CA develop psychopathology (Greif Green et al., 2010; Kessler et al., 2010). Some remain mentally healthy, succumb shortly but recover quickly, recover in the longer term, or even grow mentally after CA (Bonanno et al., 2012; Mancini & Bonanno, 2009; Masten, 2011; Rutter, 2006, 2013). These individuals may possess or acquire skills and resources that help them to adapt effectively after CA, a phenomenon known as resilience (American Psychological Association, 2016; Kalisch et al., 2017; Masten, 2011; Rutter, 2013). A better understanding of what sets these individuals apart is critically important for interventions aimed to increase resilience in those with a history of CA.

Resilience is an adaptive process following adversity, and can only be scrutinized when risk has been present (Fergus & Zimmerman, 2005; Kalisch et al., 2015; Mancini & Bonanno, 2009; Masten, 2001; Rutter, 2006, 2013; Ungar, 2015; World Health Organization, 2004). Moreover, resilience should be considered as a dynamic and changing concept, not as a static trait (Aburn, Gott, & Hoare, 2016; Afifi & MacMillan, 2011; American Psychological Association, 2016; Fergus & Zimmerman, 2005; Kinard, 1998; Luthar & Cicchetti, 2000; Masten, 2001, 2011; Masten, Best, & Garmezy, 1990; Rutter, 2013, 2006; Southwick et al., 2014; Ungar, 2013, 2015). Finally, given that resilient functioning waxes and wanes, it can be improved by resilience enhancing factors (RFs; Afifi & MacMillan, 2011; Fergus & Zimmerman, 2005; Masten, 2011; Rutter, 1985, 2013; Zolkoski & Bullock, 2012).

RFs have a promotive impact on the adjustment process following CA and thus help individuals to adapt and recover from the sequelae of CA (Rutter, 1985, 2013; Zolkoski & Bullock, 2012). Statistically, RFs operate as a moderator (Fergus & Zimmerman, 2005; Rutter, 1985), and/ or as a positive mediator (Masten, 2001; van Harmelen et al., 2016) for the relationship between CA and psychopathology. A moderating RF will operate by lowering the level of psychopathology more in adolescents with CA, compared to adolescents without CA. A mediating RF will mitigate the relationship between CA and psychopathology; if the relationship between CA and the RF has the same directionality as the relationship between the RF and psychopathology, improving the level of the RF would lower the level of psychopathology. To date, some reviews provided overviews of potential RFs (Afifi & MacMillan, 2011; Marriott, Hamilton-Giachritsis, & Harrop, 2014; Traub & Boynton-Jarrett, 2017; Wright, Kelsall, Sim, Clarke, & Creamer, 2013). Yet, these reviews were not specific to adversity in

childhood (Wright et al., 2013), examined one type of CA (e.g. childhood sexual abuse; Afifi & MacMillan, 2011; Braithwaite, O'Connor, Degli-Esposti, Luke, & Bowes, 2017; Marriott et al., 2014), examined one type of psychopathology (e.g. posttraumatic stress disorder; Braithwaite et al., 2017; Wright et al., 2013), and/ or were not conducted systematically (Traub & Boynton-Jarrett, 2017). Therefore, this is the first systematic RF review that incorporates various forms of CA and various types of psychopathology. Given that adolescence and young adulthood are characterized by a heightened risk for psychopathology (World Health Organization, 2012), I focused our review specifically on those RFs that benefit mental health in young people.

### 2.1.1 Rationale

This preregistered systematic review is aimed at offering researchers and health care providers a comprehensive overview of RFs that improve resilience to psychopathology in young people after CA. Moreover, this review may aid the development of novel resilience theories and enhance our understanding of the complex concept of resilience factors.

### 2.1.2 Objective

We aimed to identify empirically-supported RFs that reduce the risk of psychopathology in young people subsequent to CA. We focused on social, emotional, cognitive and behavioural RFs, as these factors are amenable to modification, and can be targeted in therapeutic and preventative interventions (Afifi & MacMillan, 2011; Kinard, 1998).

## 2.2 METHODS

### 2.2.1 Protocol and Registration

On the 30th of November 2016 I preregistered our review protocol at https://www.crd.york.ac.uk/PROSPERO/display_record.php?ID=CRD42016051978 (Fritz, de Graaff, Caisley, van Harmelen, & Wilkinson, 2016), to enable the reader to compare the suggested with the eventually conducted reviewing procedure.

### 2.2.2 Information Sources and Search Strategy

We searched English, Dutch and German literature in Web of Science, PsycINFO and Scopus (e.g. including MEDLINE), for all years until November 2016. Search terms, searched documents and database specific search strategies can be found in Table 2.1.

### 2.2.3 Study Selection

Duplicates were filtered out using the Mendeley reference manager. Three reviewers (Anne de Graaff, Helen Caisley and I) pilot-screened 300 titles and abstracts in November 2016. The remaining articles were screened by two of the three reviewers with an approximately equal number of articles per pair. All articles were screened based on the PI(C)OS concept (Liberati et al., 2009): Population (P),

intervention (I; i.e. RF), outcome (O), and study design (S). When P, I and O were met and the design was unknown, the full-text articles were screened for design. Incongruent ratings were solved through discussion, if necessary including a third author (Paul Wilkinson).

Table 2.1
*Used search strategy for the databases: Web of Science, Scopus and PsycINFO.*

| | **Search Terms** |
|---|---|
| | ***Search Category: Title, Abstract, & Keywords*** |
| | (resilien* OR advers*) |
| AND | (child* OR infan* OR adolescen* OR teen* OR youth* OR pediatr* OR paediatr*) |
| AND | ("self harm*" OR *suicid* OR psychopatholog* OR psycholog* OR psychiatr* OR emotion* OR affect* OR mental* OR disorder*) |
| | ***Search Category: Title*** |
| AND | (resilien* OR protect* OR support* OR adapt* OR promot* OR moderat* OR mediat* OR predict*) |
| AND | (advers* OR "at risk" OR hardship* OR loss* OR "family discord" OR parent* OR trauma* OR traged* OR "chronic* *stress*" OR "life *stress*" OR abus* OR maltreat* OR mistreat* OR assault* OR violen* OR molest* OR neglect*) |
| | **Searched Documents** |
| Types*[1] | (in press) articles, proceedings, conference papers, editorial materials and electronic collections |
| | **Database Specific Strategies** |
| Scopus | We searched the subject areas 'Health Sciences' (covering MEDLINE) and 'Social Sciences & Humanities' |
| PsycINFO | We additionally utilized subject headings for the two superordinate concepts: 'resilience' and 'childhood adversity': ("Resilience (Psychological)" OR "Protective Factors" OR "Adaptability (Personality)" OR "Adjustment" OR "Coping Behavior" OR "Emotional Adjustment" OR "Adaptive Behavior") AND ("At Risk Populations" OR "Risk Factors" OR "Dysfunctional Family" OR "Emotional Trauma" OR "Trauma" OR "Chronic Stress" OR "Emotional Abuse" OR "Child Neglect" OR "Verbal Abuse" OR "Child Abuse" OR "Sexual Abuse" OR "Physical Abuse" OR "Violence" OR "Domestic Violence" OR "Exposure to Violence" OR "Social Deprivation"). |

*Note.* *[1]We included all of the mentioned document types available for the three databases.

### 2.2.4 Study Selection Screening: Eligibility Criteria I

**CA.** CA, prior to age 18, was defined as one or multiple adversities including: Loss of a significant other, discord within the family, poor parenting, traumatic life events/ tragedy, chronic or life stress, hardship, at-risk environment, childhood abuse/ maltreatment/ mistreatment, and/ or childhood neglect (Dunn et al., 2011; Greif Green et al., 2010; Kessler et al., 2010). As we expect financial adversity to be indirectly related to psychopathology, via emotional adversity, we did not include financial adversity as CA (Martikainen, Bartley, & Lahelma, 2002; World Health Organization, 2009).

**RFs.** Inclusion criteria: The RF (a) is a direct effect, moderator and/ or a mediator for the relationship between CA and psychopathology, (b) belongs either to the individual-, family-, or community-level category, and (c) belongs to the cognitive, behavioural, social, and/ or emotional functioning domain. Exclusion criteria: The RF is defined (a) as financial advantage, (b) as no re-victimization, (c) as inverse of CA, (d) as inverse of psychopathology, or is (e) not amenable.

**Psychopathology.** Psychopathology was defined as general mental distress, as self-harm behaviour, as suicidal ideation, or as categorical diagnosis or continuous symptoms of any disorder included in the Diagnostic and Statistical Manual of Mental Disorders IV Text Revision (DSM-IV-TR; American Psychiatric Association, 2000).

**Design.** We included all longitudinal studies in which the RF was assessed before psychopathology, and CA was measured no later than the RF (i.e. cohort designs). Additionally, we excluded experimental designs which involved intervention on the RF.

### 2.2.5 Study Selection Rescreening: Eligibility Criteria II

The first screening led to more than 200 eligible articles. Therefore, we applied two additional selection criteria outlined below. Anne de Graaff and I rescreened the eligible articles in full-text, including the two additional selection criteria (see Figure 2.1; eligibility stage), which reduced the number of studies to a manageable number of 22 studies.

**RFs.** RFs should operate as moderator and/ or mediator for the relationship between CA and psychopathology, as this indicates that the RF is specific to CA. When the RF is a direct effect, the RF may operate the same for the whole population and may not be altered by CA. I believe that this criterion is crucial, as it ensures that our "resilience factor" definition precisely matches our "resilience" definition, i.e. good mental health despite a history of adversity. In the case of mediation, if CA predicts a potential RF positively (e.g. high rumination), then a high level of this potential RF would have to predict psychopathology positively (e.g. high rumination leads to higher psychopathology). This means that a low level of this factor (e.g. low rumination) would be referred to as RF. Similarly, if CA predicts a potential RF negatively (e.g. low cognitive reappraisal), then a high level of this potential RF would have to predict psychopathology negatively (e.g. high cognitive reappraisal leads to lower psychopathology). Hence, a high level of this factor (e.g. high cognitive reappraisal) would then be referred to as RF. Thus, especially for adolescents with CA it would be advantageous to reduce the levels of low RFs (e.g. rumination) and to enhance the levels of high RFs (e.g. cognitive reappraisal), to subsequently lower psychopathology levels. In the case of moderation, lower levels of low and higher levels of high RFs reduce psychopathology levels more in adolescents with CA, compared to adolescents without CA. Hence, according to this criterion all RFs are altered by a history of CA.

**Psychopathology.** Psychopathology had to be assessed at a mean age of 13 to 24 years. This criterion is important to enable the systematic selection of more homogeneous studies, to ease and enhance the comparability of findings across studies. We chose this age range, because it is characterized by a heightened risk for psychopathology and thus allows for relevant and insightful conclusions (World Health Organization, 2012).

### 2.2.6 Mediation Effects

The "eligibility criteria II" state that the RF must function as moderator and/ or mediator for the relationship between CA and psychopathology. Yet, when referring to mediation effect I mean "positive mediation" effects, as "negative mediation" effects do not function as RFs. More specifically, when I refer to RFs that have been supported by mediation analyses, I exclusively refer to factors that operated as "positive mediators" – i.e. their relationships with both CA and psychopathology are in the same direction (i.e. either both are negative, or both are positive, as described in section 2.5.1). A "negative mediator" would have opposite relationship directionalities with CA and psychopathology (i.e. one positive and one negative relationship), and therefore cannot function as an RF. Moreover, when I refer to a supported mediation effect, we expect that the association between CA and psychopathology is not significantly negative, as the mediator otherwise can also not function as an RF.

### 2.2.7 Data Extraction and Quality Assessment

The data extraction form was based on the STROBE report (2007) and an adapted version of Downs and Black's (1998) validated scale was used for the study quality ratings (see item templates in Appendix A.2 and A.3). Anne de Graaff and I conducted the data extraction pilot (3 studies: *M* Byrt's kappa = 0.56, *SD* = 0.29, range: 0.29 – 0.86; see Appendix A.1.1), the final data extraction (*M* Byrt's kappa = 0.74, *SD* = 0.17, range: 0.43 – 0.96; see Appendix A.1.1), and the study quality ratings (*M* Byrt's kappa = 0.61, *SD* = 0.19, range: 0.30 – 1.00; see Appendix A.1.1). Incongruent ratings were solved through consensus, if necessary including a third author (Paul Wilkinson). When articles lacked relevant information, we emailed the corresponding authors. Moreover, to be able to systematically judge the quality of the reviewed moderation and mediation analyses, Paul Wilkinson and I additionally applied quality criteria to the analysis methods (i.e. adequacy of sample size, single versus multiple RF model, quality of moderation/ mediation analysis; see Appendix A.4). Incongruent ratings were solved through consensus. Notably, the ratings of the analysis methods were not part of the pre-registered protocol and should therefore be considered as post hoc evaluation.

### 2.2.8 Data Synthesis Method

Given that we conjectured to find a heterogeneous set of eligible studies (i.e. in terms of CA, RFs, and psychopathology) a quantitative meta-analysis would not be appropriate. Therefore, a narrative synthesis was conducted.

### 2.2.9 Narrative Description of Moderating and Mediating RFs

I shall describe moderation effects as follows: "the association between CA and psychopathology is weaker for adolescents with a higher (or lower) level of the RF". I shall describe positive mediation effects as "a *high level of x* mediates the effect between CA and PP". This means that a high level of CA is associated with a high level of x and a high level of x is in turn associated with a high level of psychopathology. Hence, a *low level of x* is the RF. On the other hand, if a *low level of x* mediates the effect between CA and PP, a *high level of x* is the RF (as a high level of CA is associated with a low level of x and a low level of x is in turn associated with a high level of psychopathology).

## 2.3 RESULTS

### 2.3.1 Study Selection

After electronically removing duplicates (1139 of the initial 3108 studies, see Figure 2.1), all 1969 remaining studies were screened based on title and abstract screening, according to the criteria of the study selection *screening* stage (Eligibility Criteria I). Of the 1969 studies we identified 82 as additional duplicates or empty records (which have not been identified electronically), resulting in 1887 potential studies. Of those 1887 studies 1379 did not meet the screening criteria (Eligibility Criteria I). The exclusion of these 1379 studies, resulted in 508 remaining potential studies. Of those 508 studies 182 met the eligibility criteria of stage 1. Yet, the remaining 326 studies (508-182) had to be screened in

full-text, as for those studies we could not assess the design criterion only based on the title and the abstract. Of those 326 we could exclude 288 studies, resulting in 38 potentially eligible studies. Therefore, after initial screening we revealed 182 (508 – 326) potential studies which did not have to be screened in full text for the design criterion, plus 38 (326 – 288) potential studies that had to be screened in full text for the design criterion, resulting in total in 220 potentially eligible studies. Accordingly, those 220 studies were then rescreened in full text according to both the criteria of the study selection *screening* (Eligibility Criteria I) and the study selection *rescreening* (Eligibility Criteria II) stages. Of those 220, 198 studies could be excluded and 22 studies were thus eligible for data abstraction (Table 2.2).

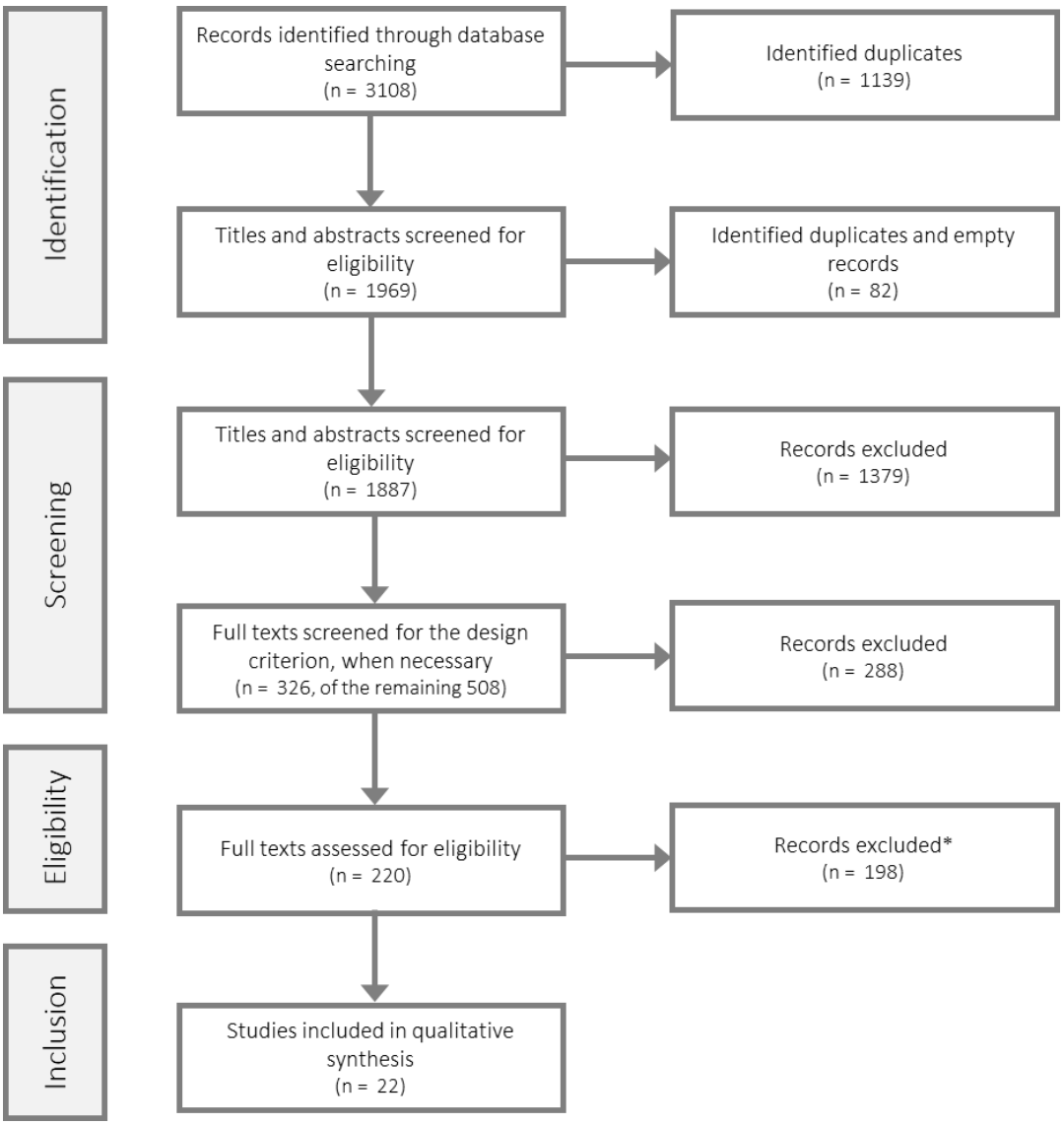



*Figure 2.1.* Study selection flow chart. We identified 878 potentially eligible studies in Web of Science, 1050 in Scopus and 1180 in PsycINFO. *Of the 198 excluded articles of the eligibility review stage, one study was identified as duplicate and three studies were excluded due to insufficient information. The Flow Chart was modelled along the PRISMA recommendations (being under a Creative Commons Attribution License; see e.g. Liberati et al. (2009), PLoS Med, can be retrieved from: https://doi.org/10.1371/journal.pmed.1000100.

### 2.3.2 Study Characteristics

All 22 studies were published in English, which is representative as only a negligible number of the screened articles were written in German or Dutch. Twenty-one of the studies included both genders (*M* male = 47.95%, *SD* = 8.27, range: 32 – 69%; see Appendix A.1.2). Walter and colleagues (2010) included females only. The studies had a mean of 3.41 time points (*SD* = 1.65, range: 2 – 9), with a time frame ranging from 10 weeks to 16 years (*M* years = 4.55, *SD* = 4.37; see Appendix A.1.2). Sample sizes ranged from 59 to 6780 participants (*M* = 1052, *SD* = 1436; see Appendix A.1.2). As shown in Figure 2.2, 27.27 percent of the studies investigated more than 1500 participants, 9.09 percent more than 1000 participants, 13.64 percent more than 500 participants, and 50 percent fewer than 500 participants. Importantly, one of the 13 studies that conducted moderation analyses had a sample size below 77, which may be insufficient in terms of power. We used a sample size of 77 as guideline, as this is the sample size that is required for moderation analyses to detect a moderate effect ($f^2$ = .15, power = .80, α = .05; see Appendix A.5). However, all 12 studies that performed mediation analyses had sample sizes higher than 150, which we assume to be sufficient in terms of power. We used a sample size of 150 as guideline, as MacKinnon, Fairchild and Fritz (2007) report that a sample size of 100 to 200 was sufficient even for multiple mediator models. At the CA assessment, the participants' mean age was 14.75 years (*SD* = 3.25, range: 11 – 22; see Appendix A.1.2). Four studies utilized a low, three a medium and two a high socio-economic status (SES) sample. Thirteen studies lacked information or did not provide an interpretation for SES. Twelve studies were performed in the United States or Canada, three in Europe, three in Israel and/ or Palestine, two in Australia, one in Korea, and one lacked information.

In total, 15 types of CAs were assessed (Appendix A.6): Five types of childhood maltreatment (nine studies), seven types of intra-family adversity (seven studies), two types of community adversity (four studies) and one clustered type of adverse life experiences (two studies). Moreover, five types of disorders and four clustered types of psychopathology have been assessed (Appendix A.6), with a mean of 1.59 assessed types of psychopathology per study (*SD* = 0.80, range: 1 – 3). Overall, 46 RFs were examined (Table 2.3), with a mean of 2.09 RFs per study (*SD* = 1.23, range: 1 – 6).

Table 2.2
*Characteristics of the Analysed Studies*

**A: Sample Characteristics**

| S | Gender | Analysis *N* | *M**[1] age | T for gender | % male | SES level | Nationality |
|---|---|---|---|---|---|---|---|
| (Banducci, Lejuez, Dougherty, & MacPherson, 2017) | both | 244 | 12 | T2 (baseline) | 54.5 | - | US |
| (Boyes, Hasking, & Martin, 2015) | both | 1973 | 14 | T1 | 32 | high | Australia |
| (Cui & Conger, 2008) | both | 451 | - | T1 | 47.67 | - | US |
| (Dennison et al., 2016) | both | 59 | 17 | T1 | 39 | - | US |
| (Dubow et al., 2012) | both | 1501 | - | T1 | 49.24 | - | Palestine & Israel |
| (Finan, Schulz, Gordon, & McCauley Ohannessian, 2015) | both | 492 | 16 | T1 | 47.5 | - | US |
| (Gaté et al., 2013) | both | 163 | 12 | T2 | 50 | - | Australia |
| (Hankin, 2005) | both | 652 | 19 | T2 | 32.2 | - | - |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| (Hardaway, Sterrett-Hong, Larkby, & Cornelius, 2016) | both | 312 | 14 | i-sample | 50 | low | US |
| (Hébert, Cénat, Blais, Lavoie, & Guerrier, 2016) | both | 6780 | - | T1 | 42.2 | - | Canada |
| (Jester, Steinberg, Heitzeg, & Zucker, 2015) | both | 1064 | - | T1 | 69 | - | US |
| (Klasen et al., 2015) | both | 1643 | 14 | T1 (i-sample) | 49.4 | medium | Germany |
| (Lansford et al., 2006) | both | 585 | - | T1 | 52 | - | US |
| (Oshri, Rogosch, & Cicchetti, 2013) | both | 400 | - | T1 | 59.25 | low | US |
| (Qouta, El-Sarraj, & Punamäki, 2001) | both | 83 | 11 | T2 | 48.8 | low | Palestine |
| (Shahar & Henrich, 2015) | both | 332 | - | T1 (i-sample) | 45 | - | Israel |
| (van Harmelen et al., 2016) | both | 771 | - | i-sample | 41.8 | high | UK |
| (Walter et al., 2010) | female | 360 | 22 | T1 | 0 | low | US |
| (Masten et al., 1999) | both | 189 | - | i-sample | 43.4 | medium | US |
| (Calvete, 2014) | both | 1052 | 14 | i-sample | 52.6 | medium | Spain |
| (Hicks et al., 2014) | both | 2021 | 12 | T1 | 49 | - | US |
| (You & Lim, 2015) | both | 2013 | - | i-sample | 52.4 | - | Korea |

**B: Methodological Characteristics**

| **S** | **CA**[*2] | **CA measure** | **RF**[*2] | **RF measure** | **PP**[*2] | **PP measure** |
|---|---|---|---|---|---|---|
| (Banducci et al., 2017) | emotional abuse | quest. | distress tolerance | task | anxiety symptoms | quest. |
| (Boyes et al., 2015) | adverse life experiences | quest. | expressive suppression<br>cognitive reappraisal<br>rumination | quest.<br>quest.<br>quest. | psychological distress | quest. |
| (Cui & Conger, 2008) | marital distress/conflict | quest. + task*[3] | positive parenting | quest. + task*[3] | poor emotional well-being<br>externalizing<br>internalizing | quest.<br>quest.<br>quest. |
| (Dennison et al., 2016) | physical, sexual abuse | quest. + interview | behav. reward reactivity<br>emotion. reward reactivity | task<br>task*[5] | depressive symptoms | interview |
| (Dubow et al., 2012) | ethnic-political conflict | quest. | academic grades<br>self-esteem<br>positive parenting | interview<br>interview<br>interview | PTS symptoms | interview |
| (Finan et al., 2015) | parental problem drinking | quest. | family cohesion<br>adolescent-mother com.<br>adolescent-father com. | quest.<br>quest.<br>quest. | externalizing | quest. |
| (Gaté et al., 2013) | aggressive parenting | task | rumination | quest. | depressive symptoms | quest. |
| (Hankin, 2005) | emotional, sexual, physical abuse | quest. | negative cognitive style<br>insecure attachment | quest.<br>quest. | depression symptoms<br>anxiety symptoms | quest.<br>quest. |
| (Hardaway et al., 2016) | community violence | quest. | extended family support<br>parental involvement | quest.<br>quest. | internalizing<br>externalizing | quest.<br>quest. |
| (Hébert et al., 2016) | sexual abuse | quest. | maternal support | quest. | mental health problems | quest. |
| (Jester et al., 2015) | parental violence | quest. | coping expectancy<br>enhancement expectancy | quest.<br>quest. | peak alcohol use<br>heavy episodic drinking | quest.<br>quest. |
| (Klasen et al., 2015) | parental mental health problems | quest. | self-efficacy<br>family climate<br>social support | quest.<br>quest.<br>quest. | depressive symptoms | quest. |
| (Lansford et al., 2006) | physical abuse | interview | proactive parenting | interview | internalizing<br>externalizing | quest.<br>quest. |
| (Oshri et al., 2013) | emotional, sexual, physical abuse, neglect | objective | ego under vs. over-control<br>over-control vs. resilient<br>under-control vs. resilient | quest.*[6]<br>quest.*[6]<br>quest.*[6] | subtance use: cannabis<br>subtance use: alcohol<br>internalizing<br>externalizing | interview<br>interview<br>quest.<br>quest. |
| (Qouta et al., 2001) | ethnic-political conflict | quest. | mental flexibility | task | emotional disorders*[7]<br>emotional disorders*[8]<br>PTS symptoms | quest.<br>quest.<br>interview |
| (Shahar & Henrich, 2015) | ethnic-political conflict (i.e. rocket attacks) | quest. | school personnel support<br>friend support<br>immediate family support | quest.<br>quest.<br>quest. | violence commission<br>anxiety symptoms<br>depressive symptoms | quest.<br>quest.<br>quest. |
| (van Harmelen et al., 2016) | accumulated family adversity | interview | immediate family support<br>friendship support | quest.<br>quest. | depressive symptoms | quest. |
| (Walter et al., 2010) | emotional, sexual, physical abuse | quest. | protective self-cognitions*[4] | quest. | PTS symptoms | interview |
| (Masten et al., 1999) | adverse life experiences | quest. + interview | parenting quality | quest.*[6] | conduct | quest. + interview |

| | | | | | | |
|---|---|---|---|---|---|---|
| (Calvete, 2014) | emotional abuse | quest. | disconnection/ rejection<br>impaired autonomy<br>other-directedness | quest.<br>quest.<br>quest. | social anxiety symptoms<br>depressive symptoms | quest.<br>quest. |
| (Hicks et al., 2014) | stressful family-level life events | interview | socialization<br>boldness<br>prosocial peers<br>academic engagement<br>parent-child relationship<br>antisocial peers | quest.<br>quest.<br>quest.<br>quest.<br>quest.<br>quest. | substance abuse | interview |
| (You & Lim, 2015) | emotional, physical abuse, emotional, physical neglect | quest. | aggression | quest. | violent delinquency<br>non-violent delinquency | quest.<br>quest. |

*Note.* CA = childhood adversity; RF = resilience enhancing factor; PP = psychopathology; S = study; T = assessment time point; i-sample = investigated sample; quest = questionnaire; behav. = behavioural; emotion. = emotional; com. = communication; PTS = posttraumatic stress; *[1]for CA assessment; *[2]information regarding the assessment instruments can be requested from the author; *[3]observer ratings on task performance; *[4]this study contained interventions, but given that we did not expect the interventions to have an effect on the RF, we included the study; *[5]subjective ratings integrated in task; *[6]questionnaires completed by counsellors/ interviewers; *[7]self report; *[8]multiple reporters.

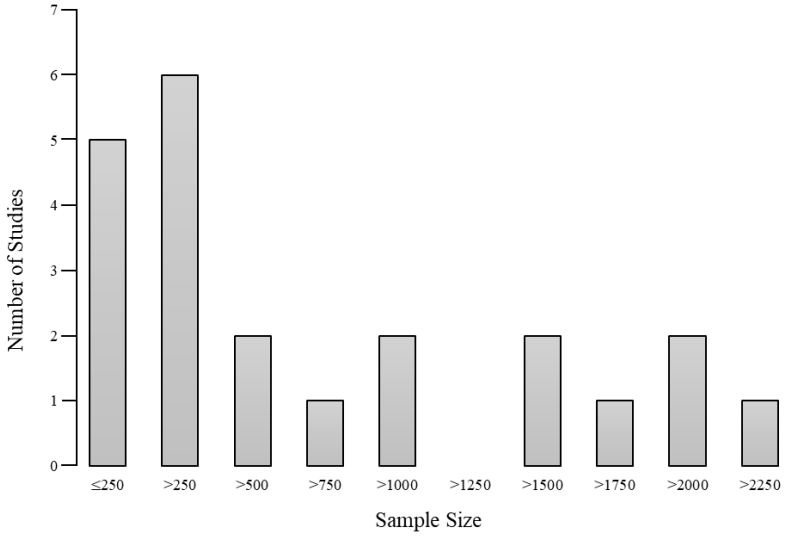



*Figure 2.2.* Sample size histogram. The histogram depicts the frequency of the studied sample sizes. The x-axis indicates the size of the studied sample in steps of 250 participants. The y-axis indicates the frequency of studies that investigated the belonging sample size.

### 2.3.3 Individual-level RFs

I report findings of individual-level RFs (Table 2.3) within four clusters. In total we found 13 supported individual-level RFs including three cognitive, four emotion regulation, three social interaction/attachment and three personality/self-concept RFs:

**Cognition and academic performance.** Qouta and colleagues (2001) found that the positive relationship between traumatic events (i.e. ethnic-political conflict) and emotional disorders (i.e. internalizing and externalizing symptoms) was stronger for adolescents with lower levels of mental flexibility (moderation). Yet, mental flexibility did not moderate the relationship between traumatic events and posttraumatic stress symptoms (Qouta et al., 2001). In the study of Boyes and colleagues (2015) the association between a history of adverse life events and psychological distress was weaker for adolescents who reported more cognitive reappraisal (moderation). Low cognitive reappraisal also mediated the association between a history of adverse life events and psychological distress (Boyes et al., 2015). Similarly, Boyes and colleagues (2015) revealed that high rumination mediates the

association between a history of adverse life events and psychological distress. However, no moderation effect was found for rumination (Boyes et al., 2015). Gaté and colleagues (2013) found that rumination does not mediate the association between aggressive parenting and depressive symptoms. Moreover, Hankin (2005) reported that a negative cognitive style no longer mediates the relationship between emotional abuse and subsequent depressive symptoms, when controlling for negative life events and an insecure attachment style. Hankin (2005) did not investigate mediation effects for other combinations of CA (i.e. sexual, physical, and/ or emotional abuse) and psychopathology (depressive or anxiety symptoms), as pairwise associations between variables were lacking. For the same reason, Hicks and colleagues (2014) did not analyse the mediation effect of academic engagement along the relationship between stressful life events and substance abuse. Finally, Dubow and colleagues (2012) found that academic grades do not moderate the association between ethnic-political conflict (e.g. violence) and posttraumatic stress symptoms. In sum, we found support for high mental flexibility, high cognitive reappraisal, and low rumination as RFs.

**Emotion regulation.** Banducci and colleagues (2017) found that adolescents with less distress tolerance and more emotional abuse experienced the most anxiety symptoms in the long term (moderation). Along these lines, Boyes and colleagues (2015) revealed that high expressive suppression mediates the association between a history of adverse life events and psychological distress, however, no moderation effect was found. In the study of You and Lim (2015), high aggression mediated the association between abuse (emotional and physical) and violent as well as non-violent delinquency. High aggression also mediated the association between emotional neglect and violent delinquency, as well as between physical neglect and non-violent delinquency. However, aggression did not mediate the association between emotional neglect and non-violent delinquency, as well as between physical neglect and violent delinquency (You & Lim, 2015). Jester and colleagues (2015) showed that high alcohol coping expectancy, i.e. consuming alcohol to handle stress, mediates the association between inter-parent violence and both peak alcohol use and heavy episodic drinking (when taking distress as intermediate predictor into account). In contrast, no mediation effects were found for alcohol enhancement expectancy, i.e. consuming alcohol to improve mood (Jester et al., 2015). Finally, Dennison and colleagues (2016) found that emotional and behavioural reward reactivity did not moderate the relationship between childhood maltreatment (physical and/ or sexual abuse) and subsequent depressive symptoms. Hence, high distress tolerance, low expressive suppression, low aggression, and low alcohol coping expectancy were supported as RFs.

**Attachment and social interactions.** Hankin (2005) found that high insecure attachment mediates the relationship between emotional abuse and depressive symptoms. No mediation effects were analysed for other combinations of CA (i.e. sexual, physical, and/ or emotional abuse) and psychopathology (i.e. depressive or anxiety symptoms), due to the lack of pairwise associations (Hankin, 2005). Calvete (2014) investigated disconnection/rejection, other-directedness and impaired autonomy factors along the relationship between two CAs (i.e. abuse by parents and peers) and two psychopathology variables (i.e. depressive and social anxiety symptoms). High disconnection/rejection mediated the relationship between abuse by peers and depressive symptoms. High other-directedness mediated the relationship between abuse by peers and social anxiety. Due to the absence of pairwise

associations, no mediation effects were analysed for other combinations of CA and psychopathology, or for impaired autonomy (Calvete, 2014). Finally, Hicks and colleagues (2014) found that socialization (e.g. obeying rules and committing to ethical values) does not mediate the relationship between stressful life events and substance abuse. Additionally, due to the absence of pairwise associations, no mediation effect was analysed for boldness (e.g. social confidence, adaptability to distress, and sensation seeking; Hicks et al., 2014). Therefore, low insecure attachment, low disconnection/rejection and low other-directedness were supported as RFs.

**Personality and self-concept.** Oshri and colleagues (2013) studied the putative RF ego control, which was split into: (a) ego over-control vs. ego resilience, (b) ego under-control vs. ego resilience and (c) ego under-control vs. ego over-control. High ego over-control vs. resilience mediated the association between early child maltreatment and internalizing, but not between early child maltreatment and cannabis use, alcohol use (see Appendix A.1.3), or externalizing behaviours. High ego under-control vs. resilience mediated the association between early child maltreatment and cannabis use, internalizing and externalizing behaviours, but not between early child maltreatment and alcohol use. For ego under-control vs. ego over-control no mediation effects were found (Oshri et al., 2013). Dubow and colleagues (2012) found that the association between ethnic-political conflict (e.g. violence) and posttraumatic stress symptoms was only significant for adolescents with a low amount of self-esteem (moderation). In contrast, in the study of Klasen and colleagues (2015) self-efficacy did not moderate the association between parental psychopathological problems and the development of depressive symptoms in the adolescent offspring. Similarly, in the study of Walter and colleagues (2010), protective self-cognitions (i.e. self-esteem and self-efficacy) did not mediate the association between child abuse and posttraumatic stress symptoms (taking resource loss as intermediate mediator into account). Thus, in the personality/self-concept cluster we found support for low ego over-control, low ego under-control, and high self-esteem.

Table 2.3
*Resilience factors tested in the analysed studies, split into individual, family, and community level*

| **Individual** | | **Family** | | **Community** | |
|---|---|---|---|---|---|
| **Supported** | **Not supported** | **Supported** | **Not supported** | **Supported** | **Not supported** |
| High distress tolerance (MO Banducci et al., 2017) | - | High positive parenting (ME Cui & Conger, 2008)+(MO Dubow et al., 2012) | Positive parenting (MO Cui & Conger, 2008) | High social support[*1] (MO Klasen et al., 2015) | - |
| High cognitive reappraisal (MO + ME Boyes et al., 2015) | - | High family cohesion (ME Finan et al., 2015) | Family cohesion (ME Finan et al., 2015) | - | Friend support (MO Shahar & Henrich, 2015)+(ME van Harmelen et al., 2016) |
| Low expressive suppression (ME Boyes et al., 2015) | Expressive suppression (MO Boyes et al., 2015) | - | Adolescent-father communication (ME Finan et al., 2015) | - | School support (MO Shahar & Henrich, 2015) |
| Low rumination (ME Boyes et al., 2015) | Rumination | - | Adolescent-mother communication | - | Prosocial peers (ME Hicks et al., 2014)[*3] |

| | | | | | |
|---|---|---|---|---|---|
| | (MO Boyes et al., 2015)+(ME Gaté et al., 2013) | | (ME Finan et al., 2015) | | |
| - | Behavioural reward reactivity (MO Dennison et al., 2016) | High extended family support (MO Hardaway et al., 2016) | Extended family support (MO Hardaway et al., 2016) | - | Antisocial peers (ME Hicks et al., 2014)[*3] |
| - | Emotional reward reactivity (MO Dennison et al., 2016) | High parental involvement (MO Hardaway et al., 2016) | Parental involvement (MO Hardaway et al., 2016) | | |
| - | Academic grades (MO Dubow et al., 2012) | - | Maternal support (MO Hébert et al., 2016) | | |
| High self-esteem (MO Dubow et al., 2012) | - | Positive family climate (MO Klasen et al., 2015) | - | | |
| Low insecure attachment (ME Hankin, 2005) | Insecure attachment (ME Hankin, 2005) | - | Proactive parenting (MO Lansford et al., 2006) | | |
| - | Negative cognitive style (ME Hankin, 2005) | High immediate family support (MO Shahar & Henrich, 2015)+(ME van Harmelen et al., 2016) | Immediate family support (MO Shahar & Henrich, 2015; van Harmelen et al., 2016) | | |
| Low coping expectancy[def1] (ME Jester et al., 2015) | - | - | Parenting quality (MO Masten et al., 1999)[*2] | | |
| - | Enhancement expectancy[def2] (ME Jester et al., 2015) | - | Parent-child relationship (ME Hicks et al., 2014)[*3] | | |
| - | Self-efficacy (MO Klasen et al., 2015) | | | | |
| Low ego over-control (ME Oshri et al., 2013) | Ego over-control (ME Oshri et al., 2013) | | | | |
| Low ego under-control (ME Oshri et al., 2013) | Ego under-control (ME Oshri et al., 2013) | | | | |
| - | Ego under- vs. over-control (ME Oshri et al., 2013) | | | | |
| High mental flexibility (MO Qouta et al., 2001) | Mental flexibility (MO Qouta et al., 2001) | | | | |
| - | Protective self-cognitions (ME Walter et al., 2010) | | | | |
| Low disconnection/ rejection (ME Calvete, 2014)[*3] | Disconnection/ rejection (ME Calvete, 2014)[*3] | | | | |
| Low other-directedness (ME Calvete, 2014) [*3] | Other-directedness (ME Calvete, 2014)[*3] | | | | |
| - | Impaired autonomy (ME Calvete, 2014)[*3] | | | | |
| - | Socialization (ME Hicks et al., 2014)[*3] | | | | |
| - | Boldness (ME Hicks et al., 2014)[*3] | | | | |

| | |
|---|---|
| - | Academic engagement (ME Hicks et al., 2014)[*3] |
| Low aggression (ME You & Lim, 2015)[*3] | Aggression (ME You & Lim, 2015)[*3] |

*Note*. MO=moderation analysis; ME=mediation analysis. [*1]Social support could potentially also include family support and should thus also belong to the family domain. [*2]The CA timeline requirements might not be fully met. [*3]The analysis did not include the direct path between CA and psychopathology when calculating the indirect mediation effect of the RF. [def1]=Consuming alcohol to handle stress. [def2]=Consuming alcohol to improve mood.

### 2.3.4 Family RFs

We split family-level RFs (Table 2.3) into two clusters and found empirical support for four family support and two parenting RFs:

**Family support.** Hardaway and colleagues (2016) found that the effect of community violence on externalizing behaviours was only significant for adolescents with a small amount of extended family support (moderation). No effect was found for the relationship between community violence and internalizing behaviours (Hardaway et al., 2016). Van Harmelen and colleagues (2016) showed that low immediate family support mediates the relationship between accumulated family adversity and depressive symptoms. No moderation effect was found (van Harmelen et al., 2016). Similarly, Shahar and Henrich (2015) revealed that immediate family support significantly attenuates the relationship between exposure to rocket attacks and both subsequent depressive symptoms and severe commission of violence (moderation). Yet, immediate family support did not moderate the relationship between exposure to rocket attacks and anxiety (Shahar & Henrich, 2015). Moreover, Finan and colleagues (2015) found that low family cohesion mediates the association between paternal alcohol abuse problems and both violation of rules (boys and girls) and aggressive conduct (girls only). No mediation effect was found for any other combination of CA (i.e. maternal or paternal alcohol abuse problems) and psychopathology (i.e. alcohol use, drug use, or binge drinking; Finan et al., 2015). Similarly, in the study of Klasen and colleagues (2015), the positive relationship between parental psychopathological problems and the development of depressive symptoms in the adolescent offspring was mitigated for adolescents who experienced a better family climate (moderation). Hence, we found support for high extended family support, immediate family support, family cohesion, and a positive family climate as RFs.

**Parental support.** Hardaway and colleagues (2016) found that the effect of community violence on externalizing behaviours was only significant for adolescents with a small amount of parental involvement (moderation). Yet, parental involvement did not moderate the relationship between community violence and internalizing behaviours (Hardaway et al., 2016). Similarly, Dubow and colleagues (2012) found that the association between ethnic-political conflict (e.g. violence) and posttraumatic stress symptoms was only significant for adolescents with a low amount of positive parenting (moderation). Cui and Conger (2008) found that low positive parenting (i.e. high positive parenting includes low negative parenting) mediates the association between marital problems and poor emotional well-being, internalizing, as well as externalizing symptoms. Moderation effects for positive parenting were mostly not supported, as only one out of 12 effects was significant (i.e. for the

association between marital distress and poor emotional well-being; Cui & Conger, 2008). Due to the absence of direct associations, Hicks and colleagues (2014) did not analyse the mediation effect of the parent-child relationship for the association between stressful life events and substance abuse. Moreover, in the study of Masten and colleagues (1999), parenting quality did not moderate the association between adverse life experiences and conduct symptoms. Similarly, Lansford and colleagues (2006) found that proactive parenting does not moderate the relationship between physical abuse and change in both internalizing symptoms and externalizing behaviours.

Two studies focussed on RFs specific to one parent (Finan et al., 2015; Hébert et al., 2016). Finan and colleagues (2015) found that adolescent-mother and adolescent-father communication (see Appendix A.1.4) do not mediate the association between parental alcohol abuse problems (i.e. maternal and paternal) and externalizing indicators (i.e. alcohol use, drug use, violation of rules, aggressive conduct, and binge drinking). Likewise, Hébert and colleagues (2016) found that maternal support does not moderate the association between childhood sexual abuse and mental health problems. Thus, in sum, parental involvement and positive parenting were supported as RFs.

### 2.3.5 Community RFs

On the community level, Klasen and colleagues (2015) found that the positive association between parental psychopathological problems and the development of depressive symptoms in the adolescent offspring is mitigated for adolescents who experienced more social support (moderation). In contrast, in the study of Shahar and Henrich (2015) school and friendship support did not moderate the relationship between exposure to rocket attacks and depressive symptoms, anxiety symptoms, as well as severe violence commission. Due to the absence of pairwise associations, van Harmelen and colleagues (2016) did not investigate the mediation effect of friendship support for the relationship between accumulated family adversity and depressive symptoms. For the same reason, Hicks and colleagues (2014) did not analyse the mediation effects of prosocial and antisocial peers along the relationship between stressful life events and substance abuse. Therefore, on the community-level high social support was supported as RF.

### 2.3.6 Single Versus Multiple RFs

Of the 22 studies, only eight have tested indirect (i.e. mediation) and/ or interaction (i.e. moderation) effects, while correcting for at least one other RF. Calvete (2014; other-directedness, disconnection/rejection, impaired autonomy), Finan and colleagues (2015; family cohesion, adolescent-mother communication, adolescent-father communication), Hankin (2005; insecure attachment, negative cognitive style), Hicks and colleagues (2014; socialization, boldness, prosocial peers, antisocial peers, academic engagement, parent-child relationship), as well as Jester and colleagues (2015; alcohol coping expectancy, alcohol enhancement expectancy) tested mediation effects, while correcting for at least one other RF. Dubow (2012; self-esteem, positive parenting, academic grades) as well as Shahar and Henrich (2015; immediate family support, school personnel support, friend support) tested interaction effects in models containing more than one RF interaction. Boyes and colleagues (2015) tested the indirect as well as the interaction effects of three RFs (expressive

suppression, cognitive reappraisal, rumination). Yet, while the mediation analysis was corrected for the respective other two RFs, in the moderation model two RFs were only entered as main effects, not as interactions (expressive suppression, rumination; Boyes et al., 2015). Hence, the current literature contains some effort to establish complex RF models that test mediation and moderation effects of RFs, while controlling for the impact of other RFs.

None of the eight mentioned studies included a model with more than six RFs. Jester and colleagues (2015) as well as Hankin (2005) first tested the indirect RF effects separately, before they performed a multiple RF model correcting for the respective other RFs. Jester and colleagues (2015) showed that alcohol coping expectancy was a significant mediator in the single and the multiple RF model, whereas alcohol enhancement expectancy was neither significant in the multiple nor in the single RF model. In contrast, in Hankin's (2005) study insecure attachment was a significant mediator in the single and the multiple RF model, whereas negative cognitive style was only a significant mediator in the single RF model. Hence, controlling for the interrelation between RFs is important as some RFs may only be significant when being tested in isolation, but not when being tested simultaneously with other individual, family, and community RFs. Along these lines, three studies found support for more than one RF in multiple RF models. This finding supports the notion that not one RF in isolation but complex interrelations of RFs affect the relationship between CA and psychopathology. In sum, such findings strongly underpin the need for a complex model that can account for various RFs following adversity, when predicting psychopathology.

### 2.3.7 Quantifying RF Effects

Comparing the effects of moderating and mediating effects statistically was not possible, as the reviewed RFs were studied following as many as 15 different forms of adversities, in the attempt to predict as many as five types of disorders (anxiety symptoms, depressive symptoms, posttraumatic stress symptoms, substance (ab)use symptoms, and conduct symptoms) and four clustered types of psychopathology (psychological distress, mental well-being, externalizing, and internalizing). Given such a variety of studied contexts, we decided that statistical comparison is not feasible. Some studies did report model related fit indices for moderation (e.g. R-squared; Klasen et al., 2015; Qouta et al., 2001) and mediation (e.g. Root Mean Square Error of Approximation (RMSEA); Cui & Conger, 2008; van Harmelen et al., 2016; Walter et al., 2010; You & Lim, 2015), but the majority of the studies did not report RF related effect sizes. The manual calculation of the effect sizes for mediating RFs might theoretically have been possible, as the proportion mediated (indirect effect divided through the total effect) could be calculated (MacKinnon et al., 2007). Yet, the interpretation of the proportion mediated is conditional on the total effect (i.e. a small proportion mediated of a large total mediation effect might with regard to actual effect still be strong, while a large proportion mediated of a small total mediation effect might with regard to the actual effect still be weak). Given that the total effects of the studies, being based on 15 different independent adversity variables and nine different dependent psychopathology variables, are so numerous, the proportion mediated would not have been comparable between studies. Moreover, the proportion mediated is only robust for sample sizes of 500 or larger (MacKinnon et al., 2007), which would only have been the case in seven studies (namely,

Boyes et al., 2015; Calvete, 2014; Hankin, 2005; Hicks et al., 2014; Jester et al., 2015; van Harmelen et al., 2016; You & Lim, 2015), of which three are statistically controversial as they lack the impact of the direct effect (namely, Calvete, 2014; Hicks et al., 2014; You & Lim, 2015). Similarly, we considered the calculation of effect sizes for moderation RFs as not feasible. Firstly, standard effect sizes such as the incremental $R^2$, which indicates the contribution of an interaction to the moderation model, are difficult to interpret, as they merely designate the contribution of an interaction and not the magnitude of its effect (Champoux & Peters, 1987). Moreover, for more advanced calculations of effect sizes the necessary information, such as the Mean Square Residuals (MSR; Bodner, 2017), was not provided.

### 2.3.8 Study Quality

**Reporting, internal and external validity.** Individual quality items were met by a mean of 16 studies (Figure 2.3; *SD* = 6.97, range: 2 – 22). The quality item "adjustment for variability in follow-up length between participants" (item 13; Downs & Black, 1998) was the least frequently met item, being met by only two studies (Dennison et al., 2016; Qouta et al., 2001). Similarly, the item assessing whether the researchers who measured psychopathology were in experimental terms blind (item 11), was only met by three studies (Boyes et al., 2015; Calvete, 2014; Oshri et al., 2013). In contrast, as much as eight quality rating items (items 1, 2, 12, & 14-18) were met by all studies. Those eight included for example the items "clarity of study aim" (item 1) or "sufficient description of the psychopathology variable" (item 2). Overall, all studies met more than half of the assessed quality items. Therefore, we concluded that all studies were of sufficient quality to be included (*M* = 14.55, *SD* = 2.04, range: 11 – 18).

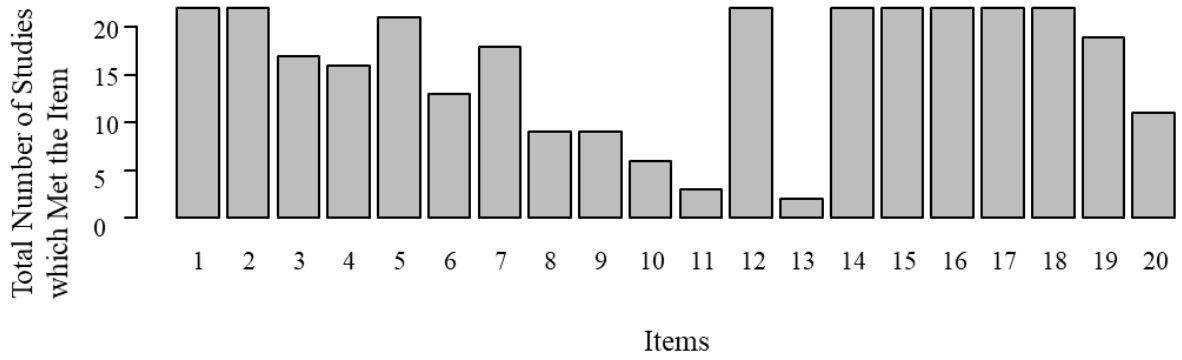



*Figure 2.3.* Quality rating distribution. The Number of Studies (y) Which Met the Respective Item of the Adapted Version of Downs' and Black's (1998) Quality Rating Scale (x). 1: Clarity of study aim, 2: Sufficient description of outcome(s), 3: Sufficient description of participant characteristics, 4: Presence of description of confounders, 5: Appropriate description of findings, 6: Report of variability estimates, 7: Description lost to follow-up characteristics, 8: Report of exact p-values, 9: Representativeness of recruitment cohort, 10: Representativeness of participation cohort, 11: Blinding, 12: Clarity about data dredging, 13: Adjustment for variability in follow-up length between participants, 14: Adequacy of statistical tests, 15: Accurate CA measure(s), 16: Accurate RF measure(s), 17: Accurate psychopathology measure(s), 18: Recruitment of same population for participants of different CA groups, 19: Correction for confounding, 20: Loss to follow-up correction.

**Quality of the analytic methods.** Ten studies performed moderation (five multiple regression analyses (MRAs), three growth models, two path models; Banducci et al., 2017; Dennison et al., 2016;

Dubow et al., 2012; Hardaway et al., 2016; Hébert et al., 2016; Klasen et al., 2015; Lansford et al., 2006; Masten et al., 1999; Qouta et al., 2001; Shahar & Henrich, 2015), nine mediation (one MRA, seven path models or structural equation models (SEMs), one SEM based on probit regression; Calvete, 2014; Finan et al., 2015; Gaté et al., 2013; Hankin, 2005; Hicks et al., 2014; Jester et al., 2015; Oshri et al., 2013; Walter et al., 2010; You & Lim, 2015), and three both types of analyses (four MRAs, two SEMs; Boyes et al., 2015; Cui & Conger, 2008; van Harmelen et al., 2016). Three studies did not control for the direct effect between CA and psychopathology when calculating mediation effects (Calvete, 2014; Hicks et al., 2014; You & Lim, 2015), which violates Baron and Kenny's (1986) traditional mediation approach. Moreover, in Masten and colleagues' (1999) study, parts of the CA index may have been assessed later than the RF. Hence, these four studies should be interpreted with caution.

To be able to judge the qualitative value of the moderation and mediation analyses we additionally applied quality criteria to the analysis methods (i.e. this was not part of the pre-registered protocol and should therefore be considered as post hoc evaluation). Moderation analyses received (a) a "1" when lacking correlational and significance testing for the relationship between CA and psychopathology at different levels of the moderator variable, (b) a "2" for correlational post hoc probing of the relationship between CA and psychopathology at different levels of the moderator variable, and (c) a "3" for regression analytic post hoc probing of the relationship between CA and psychopathology at different levels of the moderator variable. Detailed descriptions of these analytic methods can be found in Holmbeck (2002). Mediation analyses received (a) a "1" for either no calculation of the overall indirect effect or the usage of the "direct effect reduction to non-significance" criterion, (b) a "2" for the calculation of the Sobel test or comparable indirect effect tests, and (c) a "3" for the usage of bootstrap methods for the calculation of the indirect effect. Detailed descriptions of these analytic methods can be found in MacKinnon and colleagues (2007). The quality ratings can be found in Table 2.4.

Of the 13 studies which analysed moderation effects, one study could not be rated for its analytic quality, as it did not contain a description of whether post hoc probing would have been performed in case of significant interaction effects (van Harmelen et al., 2016). Moreover, three of the 12 studies were rated with a 1 (see Table 2.4) and the remaining nine studies with a 3. Of the 12 studies that tested mediation, one study was rated with a 1, six studies were rated with a 2, and five studies were rated with a 3. In sum, we concluded that the majority (moderation: 75%; mediation: 91.67%; total 83.34%) of the analytic methods that were used by the studies to test RFs are in line with the general guidelines for testing moderation and mediation, and can be considered as qualitatively adequate.

Splitting the results into systemic levels (i.e. individual, family and community levels) showed that for the individual level RFs 80 percent of the moderation analyses and 94.74 percent of the mediation analyses were qualitatively adequate (rating of 2 or higher). For the family level RFs 77.78 percent of the moderation analyses and 100 percent of the mediation analyses were qualitatively adequate. Similarly, for the community level RFs 66.67 percent of the moderation analyses and 100 percent of the mediation analyses were qualitatively adequate. The analytic quality was examined in percentages to control for the impact of the differing number of performed analyses on each systemic

level. Overall, we did not identify any trend regarding analytic quality differences between individual, family, and/ or community RFs.

Table 2.4
*Quality ratings for the analysis methods that were used to analyse the resilience factors, split into individual, family, and community level*

| Resilience factor | Study | Moderation quality | Moderation supported | Mediation quality | Mediation supported |
|---|---|---|---|---|---|
| **Individual** | | | | | |
| distress tolerance | (Banducci et al., 2017) | 3 | yes | NA | NA |
| cognitive reappraisal | (Boyes et al., 2015) | 3 | yes | 3 | yes |
| expressive suppression | (Boyes et al., 2015) | 3 | no | 3 | yes |
| rumination | (Boyes et al., 2015) | 3 | no | 3 | yes |
| rumination | (Gaté et al., 2013) | NA | NA | 3 | no |
| behavioural reward reactivity | (Dennison et al., 2016) | 3 | no | NA | NA |
| emotional reward reactivity | (Dennison et al., 2016) | 3 | no | NA | NA |
| academic grades | (Dubow et al., 2012) | 3 | no | NA | NA |
| self-esteem | (Dubow et al., 2012) | 3 | yes | NA | NA |
| insecure attachment | (Hankin, 2005) | NA | NA | 2 | yes |
| negative cognitive style | (Hankin, 2005) | NA | NA | 2 | no |
| coping expectancy[def1] | (Jester et al., 2015) | NA | NA | 2 | yes |
| enhancement expectancy[def2] | (Jester et al., 2015) | NA | NA | 2 | no |
| self-efficacy | (Klasen et al., 2015) | 1 | no | NA | NA |
| ego over-control | (Oshri et al., 2013) | NA | NA | 3 | yes |
| ego under-control | (Oshri et al., 2013) | NA | NA | 3 | yes |
| ego under- vs. over-control | (Oshri et al., 2013) | NA | NA | 3 | no |
| mental flexibility | (Qouta et al., 2001) | 1 | yes | NA | NA |
| protective self-cognitions | (Walter et al., 2010) | NA | NA | 1 | no |
| disconnection/rejection[*3] | (Calvete, 2014) | NA | NA | 3 | yes |
| other-directedness[*3] | (Calvete, 2014) | NA | NA | 3 | yes |
| impaired autonomy[*3] | (Calvete, 2014) | NA | NA | 3 | no |
| socialization[*3] | (Hicks et al., 2014) | NA | NA | 2 | no |
| boldness[*3] | (Hicks et al., 2014) | NA | NA | 2 | no |
| academic engagement[*3] | (Hicks et al., 2014) | NA | NA | 2 | no |
| aggression[*3] | (You & Lim, 2015) | NA | NA | 3 | yes |
| **Family** | | | | | |
| positive parenting | (Cui & Conger, 2008) | 1 | no | 2 | yes |
| positive parenting | (Dubow et al., 2012) | 3 | yes | NA | NA |
| family cohesion | (Finan et al., 2015) | NA | NA | 2 | yes |
| adolescent-father com. | (Finan et al., 2015) | NA | NA | 2 | no |
| adolescent-mother com. | (Finan et al., 2015) | NA | NA | 2 | no |
| extended family support | (Hardaway et al., 2016) | 3 | yes | NA | NA |
| parental involvement | (Hardaway et al., 2016) | 3 | yes | NA | NA |
| maternal support | (Hébert et al., 2016) | 3 | no | NA | NA |
| positive family climate | (Klasen et al., 2015) | 1 | yes | NA | NA |
| proactive parenting | (Lansford et al., 2006) | 3 | no | NA | NA |
| immediate family support | (Shahar & Henrich, 2015) | 3 | yes | NA | NA |
| immediate family support | (van Harmelen et al., 2016) | NR | no | 2 | yes |
| parenting quality[*2] | (Masten et al., 1999) | 3 | no | NA | NA |
| parent-child relationship[*3] | (Hicks et al., 2014) | NA | NA | 2 | no |
| **Community** | | | | | |
| social support[*1] | (Klasen et al., 2015) | 1 | yes | NA | NA |
| friend support | (Shahar & Henrich, 2015) | 3 | no | NA | NA |
| friend support | (van Harmelen et al., 2016) | NA | NA | 2 | no |
| school support | (Shahar & Henrich, 2015) | 3 | no | NA | NA |
| prosocial peers[*3] | (Hicks et al., 2014) | NA | NA | 2 | no |
| antisocial peers[*3] | (Hicks et al., 2014) | NA | NA | 2 | no |

*Note.* com. = communication. NA = not performed; NR = not rateable, or more specifically, no significant effect and no information provided whether post hoc tests were applied in case of significant effects (i.e. in case of nonsignificant effects, follow up post hoc probing tests are not necessary for moderation). [*1]The social support

measure could potentially also include family support and should therefore also belong to the family domain. [*2]The CA timeline requirements might not be fully met. [*3]The analysis did not include the direct path between CA and psychopathology when calculating the indirect mediation effect of the RF. [def1]Consuming alcohol to handle stress. [def2]Consuming alcohol to improve mood.

## 2.4 DISCUSSION

The aim of this systematic review was to identify empirically supported RFs that benefit mental health in young people following CA. We reviewed 22 studies, including 46 amenable RFs. Thirteen of 25 individual-level RFs, six of 12 family-level RFs, and one of five community-level RFs were found to reduce the risk of psychopathology following CA. The absolute number of supported RFs seems to indicate that individual- and family-level RFs are most effective. However, the seemingly lower relevance of community-level RFs may be artefactual due to the small number of community-level studies that we could include in this review.

The 13 supported individual-level RFs included three cognitive (high: cognitive reappraisal, mental flexibility; low: rumination), four emotion regulation (high: distress tolerance; low: alcohol coping expectancy, aggression, expressive suppression), three social interaction/attachment (low: insecure attachment, disconnection/rejection, other-directedness) and three personality/self-concept RFs (high: self-esteem; low: ego over-control, ego under-control). It is as yet unknown whether these RF dimensions have compensatory effects, in the sense that an individual who performs low on one of those dimensions might still be functioning resiliently through performing high on other dimensions. Moreover, for most of the RFs it is also unknown to what extent they overlap in their prediction of mental health resilience.

Supported family-level RFs consisted of four family support (high: family cohesion, positive family climate, immediate family support, extended family support) and two parenting RFs (high: positive parenting, parental involvement). Interestingly, all RFs that were specific to one parent, e.g. adolescent father communication or maternal support, were not supported as RFs. This may suggest that the totality of family support is more important for resilience, than the quality of support from individual family members. Yet, as for the individual-level RFs, it is unknown to what extent the RFs overlap in their prediction of mental health resilience.

The fact that on the community-level only high social support was revealed as RF might suggest that a general social network has a stronger resilience enhancing effect than specific types of social support. However, given the restricted number of included community-level studies this conclusion is rather preliminary and requires further investigation. For example, our lab recently found that friendship support predicts resilient functioning in young people (van Harmelen et al., 2017). Thus, although only one RF was revealed on the community-level, this does not suggest that community-level RFs are less important for mental health resilience. Rather, community-level RFs have had less attention than individual- and family-level RFs and therefore require further investigation. A more thorough examination of community-level RFs may enhance our understanding of the overall picture of systemic levels that benefit mental health resilience. On the whole, our review found support for RFs on all studied

systemic levels, i.e. individual-, family- and community-levels, which indicates a movement towards a more complete understanding of the resilience concept.

Despite the movement to a more systemic approach, only eight of the reviewed studies corrected for the impact of at least one other RF, when testing the indirect and/or interaction effect of an RF (i.e. multiple RF model). Findings of single versus multiple RF models indicated that taking the interrelatedness of RFs into account is important, as some RFs may only be significant when being tested in isolation, but not when being tested simultaneously with other individual, family, and/ or community RFs. Along these lines, three studies found support for more than one RF in multiple RF models. This supports the notion that not one RF in isolation, but that a multifarious set of RFs seems to affect the relationship between CA and psychopathology. Such findings strongly underpin the need for a complex model that can account for various RFs following adversity that benefit mental health resilience.

It would have been advantageous if effect sizes could have been calculated for moderation and mediation effects. This would have allowed us to draw conclusions regarding the magnitude of specific RF effects. Knowing the magnitude of RFs is beneficial, as it gives an indication about which factors might be most efficient targets. In the future, open data sharing, as was for example done by van Harmelen and colleagues (2016), may facilitate RF comparisons. Given that our findings suggest that RFs do not function in isolation, it would be advantageous to know effect sizes of isolated RF effects, yet it would perhaps be even more interesting to establish and examine the effects of several RFs being clustered in complex systems of unidirectional or directional interrelations. This would help to disentangle whether RFs are indeed interrelated and function as a protective system.

For a systematic review it is of critical importance to carefully assess and investigate the (a) reporting, (b) internal, (c) external, and (d) the analytic quality of the studies. As all studies met more than half of the assessed quality items (i.e. for reporting, internal, & external validity), we decided that all studies were of sufficient quality to be included. However, the quality ratings were not without limitations. For example, Downs and Black's (1998) quality criteria are not specific to cohort studies and some more recent statistical improvements, such as the match of the variable level and the analysis technique (e.g. categorical vs continuous data analysis methods), are not directly covered. Critics might further argue that the impact of studies in a systematic review should be weighed according to the study quality. Given that the set of reviewed studies was highly disparate and fairly incomparable, weighing according to "reporting", "internal" or "external" validity criteria would not have been insightful. Yet, as the systematic review focussed on moderating and mediating RFs, we considered it most insightful to apply weights based on the quality of the applied moderation and mediation methods. Of the studies that (a) performed moderation analysis and (b) could be rated for the analytic quality, 75 percent applied qualitatively adequate analysis techniques. Of the studies that tested mediation, 91.67 percent applied adequate analysis techniques. Therefore, we concluded that the majority (83.34%) of the applied analytic methods could be considered as qualitatively adequate. Moreover, we did not identify any trend regarding analytic quality differences between individual, family and/ or community RFs. I believe that this finding supports our conclusion that RFs are not restricted to one systemic level but are found to

function on all three investigated systemic levels. Therefore, I call future research to focus on a more systemic and complete understanding of the RF concept.

The reviewed studies were conducted in as many as eight different countries: United States [11 studies], Israel and/ or Palestine [3 studies], Australia [2 studies], Canada [1 study], UK [1 study], Spain [1 study], Germany [1 study], and in Korea [1 study]. Moreover, all 22 reviewed studies were published in English and only a negligible number of the 1969 screened studies were published in German and Dutch. Hence, research scrutinizing resilience promoting factors seems to be an international imperative. Yet it needs to be noted, that despite the variety of studied nations, mainly Western populations were studied.

Even though the studies were highly disparate, 95.45 percent of the studies researched both genders with on average 47.95 percent males per sample. Therefore, we consider the review overall as gender balanced and on average gender representative. Nine studies provided a proper SES description, which covered a range from low to high SES (4 low, 3 medium, 2 high). However, not enough studies provided sufficient information to draw a conclusion regarding the studies' representativeness of SES. Along these lines, no conclusion can be drawn whether RFs operate the same for adolescents with different SES levels. Similarly, as the studies varied strongly in the studied time frame, which ranged from 10 weeks to 16 years, and given that the CA assessment age ranged from age 11 to age 22, no conclusions are warranted regarding timing effects or critical developmental windows.

Whereas all studies that performed mediation analyses were considered to have a sufficiently large sample size, one of the 13 studies that conducted moderation analyses may have had an insufficient sample size. This moderation study failed to find significant moderation effects for the two tested RFs (emotional and behavioural reward reactivity; Dennison et al., 2016). In sum, the majority of the reviewed studies seemed to be appropriate in terms of statistical power. However, shortcomings raising the possibility of type I errors are that: (a) not all studies were underpinned by resilience-focused hypotheses (Fergus & Zimmerman, 2005), (b) some RFs were secondary findings, (c) most RFs were only significant in one study, and (d) some positive findings were not replicated with different combinations of CA and psychopathology.

Regarding the studied designs, we only included cohort designs in which the RF was assessed before psychopathology and CA was measured no later than the RF. This design criterion was of major importance, as it ensured a causal timeline according to which psychopathology at the time of the outcome assessment would less likely have affected the RF and the RF would less likely have affected the CA experience. However, a more advanced design would have been to also assess the RFs prior to the occurrence of CA, so that baseline levels of the RFs could have been taken into account. This would have allowed us to draw more stringent conclusions regarding which RFs are specific to mental health resilience after CA, and which RFs are time-independent and are predictive for mental health resilience regardless of being measured prior to or after CA. Similarly, if psychopathology would also have been measured prior to or together with CA, conclusions could have been drawn regarding the development of mental ill-health following CA, taking into account the baseline psychopathological level. Notably, some of the reviewed studies did control for baseline psychopathology levels. In sum, future

research should investigate which of the RFs that predict mental health resilience are specific to the time period after the CA experience and which RFs are time-independent. Moreover, future research should not only examine the effectiveness of RFs in reducing the risk of psychopathology following CA, but should also examine the effectiveness of RFs in reducing the risk of the development of psychopathology following CA.

Critics might further raise the concern that our review does not capture resilience dynamics, given that most of the reported studies assessed the RFs at a single point in time. This is of course correct; the effectiveness of RFs may indeed fluctuate. Based on our findings we can only draw the very general conclusion that the supported RFs seem to alter the relationship between prior CA and subsequent psychopathology.

Overall, the review should be viewed in the light of the heterogeneity of the included studies (i.e. follow-up length, sample size, CA assessment age range, CA/ RF/ psychopathology assessment method, number of CA/ RF/ psychopathology types assessed per study, applied analysis techniques). Therefore, I do not claim that the supported RFs are protective following every type of CA, for every type of psychopathology, for individuals of all cultures, or at all developmental stages. In other words, it may potentially be the case that some of the reviewed RFs are supportive in one, but not in another context. For example, low levels of expressive suppression (i.e. low levels of suppressing emotions) may be protective in safe environments, but may be ineffective or perhaps even disadvantageous in highly dangerous and hazardous environments. As we reviewed 42 different RFs following 15 different forms of CA in an attempt to predict at least one out of nine different types of psychopathology, I ask the readers to be aware that our results are based on averages and may not generalize to all contexts, especially not when those are extreme and/ or exceptional. Yet, I cautiously conjecture that the supported RFs might be potential targets for alleviating the relationship between CA and psychopathology in young people. Nonetheless, replication research is critically needed to investigate the generalizability of RFs between people and across situations.

The fact that only two reviewed RFs were significant in more than one study, additionally highlights the crucial need of replication studies. Thus, future research should (a) replicate RF findings, (b) further examine community-level RFs, (c) study RF fluctuations as well as critical windows, and (d) scrutinize the preventative and therapeutic effectiveness of RF enhancement. In sum, this is the first preregistered systematic review on social, cognitive, emotional and behavioural RFs that attenuate psychopathology in young people after CA. The review revealed evidence for 20 amenable RFs. The review provided support for a systemic framework of mental health resilience, as the identified RFs functioned on individual-, family- and community- levels. Moreover, our findings suggest that RFs may together function as interrelated, protective system. Therefore, I advocate for more research along the lines of systemic resilience theories, and for studying RFs as complex interrelations that eventually mediate and/ or moderate the relationship between CA and psychopathology. A first attempt on studying RFs as an interrelated, complex system in form of a network model will be presented in Chapter 3.

# CHAPTER 3

# A Network Model of Resilience Factors for Adolescents with and without Exposure to Childhood Adversity

Childhood adversity (CA) has been suggested to be "psychiatry's greatest public health challenge" (p. e300 in Grant & Lappin, 2017). It is often assumed that adversities are unusual and uncommon experiences (Bonanno et al., 2011), but large, population-representative research (Greif Green et al., 2010; Kessler et al., 1997, 2010) has shown that up to 53.4 percent of individuals under the age of 18 report having experienced at least one form of CA (Greif Green et al., 2010). As described in the previous chapters, CAs span a wide range of severely stressful and traumatic experiences (Greif Green et al., 2010; Kessler et al., 1997, 2010), and account for more than a quarter of all mental health problems (Kessler et al., 2010). CAs can range from one-time events such as a loss of a significant other, a severe traffic accident, or sexual assault, to chronic experiences such as emotional neglect, physical maltreatment, or parental mental illness (see Chapter 2 or Fritz, de Graaff, Caisley, van Harmelen, & Wilkinson, 2018). Given that CA poses a crucial risk to subsequent mental health problems, it is vital to examine how we can reveal and, where possible, facilitate mental health resilience in order to reduce the negative consequences of CA.

Mental health resilience describes the process of effective adaptation, i.e. staying mentally healthy, following adversity (American Psychological Association, 2016; Kalisch et al., 2017; Masten, 2011; Rutter, 2013). In other words, although CA increases the risk of mental illness, not all those exposed go on to develop mental health problems. Based on this concept, resilience factors (RFs) are defined as characteristics, skills and resources that reduce the risk of mental health problems subsequent to CA (Chapter 2; Afifi & MacMillan, 2011; Marriott et al., 2014; Traub & Boynton-Jarrett, 2017). So far, resilience factors have often been modelled as single main-, moderation-, and mediation-effects (Chapter 2; Fergus & Zimmerman, 2005; Masten, 2011). Resilience researchers have also started using growth curve models and predictive difference scores to aid the revealing and understanding of resilient functioning (Bonanno et al., 2012; Bonanno, Romero, & Klein, 2015; Bonanno et al., 2011; Masten, 2011; van Harmelen et al., 2017). However, these approaches do not take into account that there are a range of RFs that are interrelated and potentially have combined effects, although it is commonly recognized that RFs do not function in isolation from each other (Bonanno et al., 2011; Davydov, Stewart, Ritchie, & Chaudieu, 2010; Fergus & Zimmerman, 2005; Kinard, 1998; Mancini & Bonanno, 2009; Masten, 2001, 2011; Rutter, 1985; Ungar, 2013). For example, Boyes, Hasking and Martin (2015) showed that expressive suppression, cognitive reappraisal and rumination together mediate the association between a history of CA and mental distress. Crucially, no single RF has been reported as having a leading effect in benefitting mental health resilience (Bonanno et al., 2015, 2011), which supports the conjecture that mental health resilience is better represented as an interrelated system of RFs.

Here, I aim to characterize the architecture of this system of RFs and its relationship with concurrent distress, in order to enhance our understanding of the putative mechanisms of RFs that may

reduce the liability of poor mental health following CA. To this end, network analysis will be applied, a statistical methodology that estimates and scrutinizes the unique interrelations among many variables at the same time (Borsboom & Cramer, 2013; for a detailed, methodological rationale see Appendix B.1). In the last few years, network analysis has been utilized as psychometric tool for the exploration of psychopathology (Borsboom & Cramer, 2013; Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011; Cramer, Waldorp, van der Maas, & Borsboom, 2010; Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012; Schmittmann et al., 2013; Schweren, van Borkulo, Fried, & Goodyer, 2018). In the present study, my colleagues and I set out to model the interrelations of selected RFs which are derived from our recent pre-registered systematic literature review (Fritz, de Graaff, et al., 2018), which is described in Chapter 2. To the best of our knowledge, this is the first time network analysis is used to estimate network models of RFs. Of note, we do not aim to study "network-related resilience", e.g. the ability of a network to adjust flexibly to internal and external errors to remain functional (Barabási, 2016); rather, we aim to investigate how empirically-supported factors that enhance "mental health resilience" in adolescents following CA relate to each other. Thus, throughout the present chapter resilience refers exclusively to mental health resilience.

We focus on RF networks in adolescence, as this is a crucial developmental period for the first emergence of mental health problems (see Chapter 1; The Lancet, 2016). First, we shall compare and contrast RF-RF interrelations between groups of adolescents exposed to ("CA+ group") and not exposed to CA ("CA- group"). Given that adolescents exposed to CA have on average a higher vulnerability to mental health problems than adolescents not exposed to CA (Kessler et al., 2010), we assume that this heightened vulnerability may go together with lower RF levels, and may influence how RFs interrelate. Second, we shall examine the influence of a general distress factor, indexing mental health problems, on the RF network models of the CA+ and the CA- group. As the RFs included in the present study were empirically found to mitigate (i.e. moderate and/or mediate) the positive relationship between CA and mental health problems (see Chapter 2), we expect that the general distress variable may differentially influence CA+ and CA- group networks. With the suggested RF network analyses we aim to provide novel insights into the architecture of empirically supported RFs, and their relations with general distress, which may advance our understanding of the complex system of factors that improve mental health resilience.

### 3.1.1 Aims and Hypotheses

Three consecutive research aims will be studied:

1. *Estimating RF network models*: We expect that RFs will be related to each other in both group networks, but that CA+ and CA- networks will be dissimilar in structure.
2. *Estimating RF network models including a general distress index*: We will explore the impact of general distress levels on the network structures, through contrasting the CA+ and CA- network structures after adding a general distress variable to the networks.
3. *Investigating potential group differences due to the influence of general distress on the network models:* We will further scrutinize whether potential differences in CA+ and CA- networks, upon

taking distress levels into account, result from (1) corrected “RF-RF” interrelations, (2) “RF-distress” interrelations, or (3) both.

## 3.2 METHODS

### 3.2.1 Design

ROOTS is a large-scale adolescent cohort (total *N* = 1238) in which 14-year-olds from 18 schools in Cambridgeshire were assessed (UK; 2005 to 2006). Before participation the adolescents and their caregiver had to provide written informed consent. The aim of the ROOTS study was to measure risk and resilience factors, in an attempt to predict and understand the development of psychopathology (Goodyer, Croudace, Dunn, Herbert, & Jones, 2010). The study was confirmed by the Cambridgeshire Research Ethics Committee (No: 03/302) and was conducted in line with the Declaration of Helsinki as well as Good Clinical Practice guidelines.

### 3.2.2 Sample

We included all adolescents who had complete data for CA (total *N* = 1139; CA+ *n* = 638; CA- *n* = 501). The sample included 620 girls and 519 boys. The adolescents had a mean age of 14.49 years (*SD* = 0.28, range: 13.88 – 15.28; see also Table 3.1).

### 3.2.3 Childhood Adversity (CA)

CA was assessed at age 14, with the semi-structured Cambridge Early Experiences Interview (Dunn et al., 2011) being conducted with the adolescent’s main caregiver (96% maternal report). The following topics were assessed: Family loss, family discord, atypical parenting style, lack of maternal affection/engagement, periods of unemployment, financial difficulties, parental/sibling psychiatric illness, parental/sibling medical illness with impact, sexual/emotional/physical abuse, criminality amongst family members, acute social disturbances, and chronic social difficulties (Dunn et al., 2011). The interview focussed on three timeframes (early childhood (EC): 0 to 5 years; later childhood (LC): 5 to 11 years; early adolescence (EA): 11 to 14 years) with the aim to enhance recall quality and to reduce the risk of recall bias (Dunn et al., 2011). In a previous report on this sample, Dunn and colleagues (Dunn et al., 2011) clustered adolescents based on their CA experiences using latent class analysis (LCA). They revealed four CA classes: No CA (EC = 68.8%, LC = 59.3%, EA = 64.4%), moderate CA (EC = 18.7%, LC = 25.5%, EA = 21.7%), severe CA (EC = 5.8%, LC = 10.0%, EA = 6.9%), and atypical parenting CA (EC = 6.7%, LC = 5.2%, EA = 7.0%). The four latent classes revealed good class assignment accuracies, ranging from 79 to 95 percent, and the risk of psychopathological distress increased with the adversity intensity of the classes, indicating discriminant validity of the classes (Dunn et al., 2011). To ensure sufficient analytic power and consistency with previous reports on this sample, we split the adolescents in two CA groups: Group 1 in which the adolescents belonged to the no CA class for all time intervals (i.e. CA- group, 44%), and group 2 in which the adolescents belonged to a class other than no CA for at least one time interval (i.e. CA+ group, 56%).

### 3.2.4 General Distress

Depression symptoms were assessed with the Mood and Feelings Questionnaire (33 items; Messer, Angold, & Costello, 1995). Anxiety symptoms were assessed with the Revised Children's Manifest Anxiety Scale (28 items; Reynolds & Richmond, 1978). In a previous report on this sample, a bifactor model of these sixty-one items revealed one latent factor termed the general distress factor and three specific group factors (hopelessness/ suicidal thoughts, generalized worrying, and restlessness/ fatigue; Brodbeck, Abbott, Goodyer, & Croudace, 2011). Here we exclusively utilize the general distress factor, as this single measure revealed the highest measurement precision (i.e. lowest conditional standard error of measurement), and all items except two loaded well on it (Brodbeck et al., 2011). A further report showed that severe mental illness symptoms also loaded well onto the general distress factor (Stochl et al., 2015). Moreover, the general distress factor has good external validity, being replicated in two additional large-scale cohorts (St Clair et al., 2017; Stochl et al., 2015).

### 3.2.5 Resilience Factors (RFs)

ROOTS included 10 of the 20 RFs that were identified in our systematic review (which is described in Chapter 2 and in Fritz, de Graaff, et al., 2018). All RFs are assessed via adolescent self-report (unless stated otherwise). Detailed information on reliability and validity of all measures can be found in Appendix B.2.

1. High friendship support. We used five items of the Cambridge Friendships Questionnaire (Goodyer, Wright, & Altham, 1989) to assess friendship support (e.g. "Can you confide in your friends?"). Friendship support has been suggested to be a combination of moral support and companionship (Shahar, Cohen, Grogan, Barile, & Henrich, 2009). Here the RF captured both the quantity and the quality of friendship support an adolescent receives. More specifically, the quantity was measured as the degree to which the adolescent is satisfied with the number of friends and the frequency of contact. The quality was measured as the degree of understanding of each other's needs, being able to rely on each other, and feeling overall content with the friendship relationships (Goodyer et al., 1989). Higher levels of friendship support correspond to a higher level of the RF.
2. High family support. We used five items of the McMaster Family Assessment Device (Epstein, Baldwin, & Bishop, 1983) to assess family support (e.g. "In times of crisis we can turn to each other for support."). Family support was set out to capture the degree to which adolescents receive and perceive emotional support and shelter from their family members. More specifically the RF measured whether the adolescent feels secure to share his/her emotions and can openly discuss fears and concerns with a family member, as well as whether the adolescent has the option to express the need for support from a family member when times are tough, can confide in support from a family member, and generally has a trusting relationship with family members (Epstein et al., 1983). Higher levels of family support correspond to a higher level of the RF.
3. High family cohesion/climate. We used the remaining seven items of the McMaster Family Assessment Device (Epstein et al., 1983) to assess family cohesion/climate (e.g. reversed: "We

don't get along well together."). To support readability we will refer to cohesion when meaning cohesion/climate. Family cohesion was set out to capture the "team spirit" as well as the cohesiveness an adolescent associates with his/her family. To this end, this RF measured practical considerations such as whether family members spend their leisure time together, are able to make group decisions, and can cooperate and solve problems. Moreover, this RF measured considerations regarding the family climate and closeness such as whether family members feel accepted, are on the same "wavelength", and whether there is a warm atmosphere within the family (Epstein et al., 1983). Higher levels of family cohesion correspond to a higher level of the RF.

4. High positive self-esteem. We used five items of the Rosenberg self-esteem scale (Rosenberg, 1965) to assess positive self-esteem (e.g. "I was satisfied with myself."). High positive self-esteem was set out to capture the extent to which an adolescent holds an optimistic self-concept. Therefore, this RF measured items such as being content about oneself, being aware of and satisfied with one's skills and worth, having an affirmative attitude towards oneself, as well as feeling equally worthy and capable as others (Rosenberg, 1965). Higher levels of positive self-esteem correspond to a higher level of the RF.
5. Low negative self-esteem. We used the remaining five items of the Rosenberg self-esteem scale (Rosenberg, 1965) to assess negative self-esteem (e.g. "I felt that I was a failure."). Low negative self-esteem was set out to capture the extent to which an adolescent refutes a negative self-evaluation. More specifically, an adolescent with a negative self-concept would believe that he or she has little to be proud of, does not respect himself/herself much, feels generally rather incompetent, has a pessimistic attitude towards himself/herself, and considers him or herself as inferior to others. An adolescent with a low level of negative self-esteem would rather disagree and therefore score low on those self-evaluations (Rosenberg, 1965). This RF was reverse coded and therefore higher levels of low negative self-esteem correspond to a higher level of the RF.
6. Low ruminative brooding. We used five items of the Ruminative Response Scale (RRS; e.g. "I think about a recent situation, wishing it had gone better."; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) and two items of the Short Leyton Obsessional Inventory (LOI; e.g. "I kept thinking about things that I had done because I wasn't sure whether they were the right things to do."; Bamber, Tamplin, Park, Kyte, & Goodyer, 2002) to assess brooding. Brooding was set out to capture "'moody pondering', negative self-evaluative thinking (e.g., 'Why can't I handle things better?') and comparative thinking about the self (e.g. 'Why do I have problems other people don't have?')" (p. 2 in Watkins & Roberts, 2020), in a passive, abstract and rather uncontrollable manner (McEvoy et al., 2018; Treynor et al., 2003; Watkins & Roberts, 2020). This RF was reverse coded and therefore higher levels of low brooding correspond to a higher level of the RF.
7. Low reflective rumination. We used five items of the RRS (Treynor et al., 2003) to assess reflective rumination (e.g. "I go away by myself and think about why I feel this way"). Reflective rumination has been suggested to be defined as "turning inward to engage in cognitive problem

solving . . . [but when done too often] can draw one into negative cycles of thinking" (p. 256 & 257 in Treynor et al., 2003). More specifically, this RF measured the tendency to analyse, write, or prolongedly cogitate about reasons and explanations for (predominantly negative) feelings and emotions (e.g. "I analyse my personality to try to understand why I am depressed", Treynor et al., 2003). In contrast to ruminative brooding, ruminative reflection is generally less abstract and comparative, and a bit more active and analytic (e.g. analyse, write about, or go away to think about my feelings). This RF was reverse coded and therefore higher levels of low reflective rumination correspond to a higher level of the RF.

8. High distress tolerance. We used five items of the Emotionality Activity Sociability Temperament Survey (Bould, Joinson, Sterne, & Araya, 2013) to assess distress tolerance (e.g. "He/she reacts intensely when upset."; note: parent report). Generally, "distress tolerance is defined as the capacity to experience and withstand negative psychological states" (p. 83 in Simons & Gaher, 2005). This RF measured examples of low distress tolerance (or, in other words, distress intolerance), such as the tendency to get emotional, upset, and agitated easily, and to react intensely when distress and emotions pile up (Bould et al., 2013). The items were inversely scored with higher scores reflecting distress tolerance. Higher levels of distress tolerance correspond to a higher level of the RF.
9. Low aggression. We used four items of the Behaviour Checklist (11 questions based on the DSM-IV [American Psychiatric Association, 2000] criteria for conduct problems) to assess aggression (e.g. "I deliberately damaged property."; Goodyer et al., 2011). Aggression can generally be expressed in different forms, including "thoughts (e.g., hostility), emotions (e.g., anger), and behavior (e.g., verbal and physical aggression) that are intended to harm another person" or animal (p. 120 in Webster et al., 2014). Here the aggression RF mainly captured physical aggression. More specifically, the RF captured items such as being prepared to resort to violence, destroying property, threatening, and hurting others. This RF was reverse coded and therefore higher levels of low aggression correspond to a higher level of the RF.
10. Low expressive suppression. We used one item of the Antisocial Process Screening Device (Poythress et al., 2006) to assess expressive suppression (i.e. "Does not show feelings or emotions."; note: parent report). Generally, expressive suppression is defined as "a form of response modulation that involves inhibiting ongoing emotion-expressive behavior" (p. 349 in Gross & John, 2003), thereby being the opposite of showing and communicating feelings or emotions. Although we only had one item, it nevertheless directly measured the degree to which the adolescent suppresses, that is "does not show", feelings or emotions. This RF was reverse coded and therefore higher levels of low expressive suppression correspond to a higher level of the RF.

### 3.2.6 Analysis

**Variable preparation.** Firstly, we computed the ten above described RFs. Nine of the ten RFs were computed with one-factor confirmatory factor analyses (CFAs). As the items were assessed on an ordinal measurement level, we estimated the CFAs based on polychoric correlations, using the

lavaan package in R (Rosseel, 2012). All CFAs provided an acceptable fit to the respective items (for details see Appendix B.3). For expressive suppression, we used a standardized item score, as this RF was assessed with a single item. As described above, we used a general distress factor as index for concurrent mental health, which we derived from a previously published bifactor model (Brodbeck et al., 2011). Secondly, we prepared the RFs and general distress variable for the network analysis. To reduce deviations from normality we applied the nonparanormal transformation to the RFs and the general distress variable (R package: huge; Zhao, Liu, Roeder, Lafferty, & Wasserman, 2012). To meet the exchangeability assumption of permutation tests, which we used to compare the CA+ and the CA- group networks (those tests are explained in depth below), we transformed variables for the overall sample before splitting the sample into CA+ and CA- adolescents, to ensure that an RF has the same scale in the CA+ and the CA- group. Moreover, we dichotomized variables that had a substantially restricted range (i.e. expressive suppression and aggression RFs).

**Network estimation.** We estimated the network models separately for the CA+ and the CA- groups. In the visualization of the network models, the RFs are depicted as circles, called “nodes” (or “vertices”; see Figure 3.1). Nodes are connected by lines, called “edges” (or “links”). The thickness of the edges indicates to what degree RFs are related, and the color of the edges indicates the relationship sign (i.e. positive = blue, negative = red; Costantini et al., 2015; McNally, 2016). Cross-sectional networks can for example represent zero-order correlations (association network) or regularized partial correlations between RFs. The regularized partial correlation network provides information about variable interrelations after controlling for all other included variables.

Those network models estimate many interrelations, leading to the risk of false positive interrelations (Costantini et al., 2015; Epskamp & Fried, 2018; Fried & Cramer, 2017). To prevent this, we used the least absolute shrinkage and selection operator (LASSO) regularization method. The LASSO sets weak partial correlations to exactly zero, almost always resulting in a sparse network (Costantini et al., 2015; Epskamp & Fried, 2018; Epskamp, Kruis, & Marsman, 2017; McNally, 2016). We applied LASSO regularization rather than significance values, as significance levels have an arbitrary threshold as well as either the disadvantage of multiple testing problems or lower power when applying multiple testing corrections (for further explanation see Costantini et al., 2015). To obtain the interrelations between variables, we used the cor_auto function (Epskamp et al., 2012) in R that estimates the appropriate correlation type: Pearson for two continuous variables, polychoric for two dichotomous variables, and polyserial for one continuous and one dichotomous variable. In the remainder of the chapter, I report the results of the regularized partial correlation networks; results of networks representing zero-order correlations can be found in Appendix B.4.

**Network inference.** Based on the estimated RF networks, interrelatedness (or “centrality”) coefficients can be calculated, which help to interpret the results of the network model. We calculated three coefficients. *Node strength* is the sum of the interrelation values (e.g. regularized partial correlations) of a given RF with all directly related RFs (i.e. the sum of the absolute values of the RF interrelations; Costantini et al., 2015; McNally, 2016). *Expected influence* is based on the formula of node strength, but takes negative relationships between RFs into account (i.e. the sum of the relative values of the RF interrelations; McNally, 2016). *Node predictability* is defined as the amount of variance

of each RF that is explained by the directly related RFs. Node predictability is an *absolute* metric ranging from zero to 100 percent explained variance. Note, for dichotomous RFs, we based the node predictability on the normalized accuracy, instead of on the variance explained (Haslbeck & Waldorp, 2018). For a detailed discussion of these results see Appendix B.5 and B.6.

**Network stability and accuracy.** To scrutinize the robustness of the estimated network models, we examined their stability and accuracy. Accuracy can be scrutinized through calculating nonparametric bootstrap confidence intervals (CIs, 95%) for the RF interrelations. The widths of these CIs give an indication for accuracy. Stability can be scrutinized through re-calculating interrelatedness coefficients such as the node strength for sample subsets. If the node strength remains similar in the subsets, this indicates that the RF network is stable (Epskamp, Borsboom, & Fried, 2018). Accordingly, we bootstrapped the RF interrelations (i.e. accuracy) and applied a subset bootstrap on node strength and expected influence (i.e. stability), with 2000 bootstraps each. For a detailed discussion of these results see Appendix B.7.

**Sensitivity analyses.** To allow for the largest possible sample size, we based the network models on the *full-information* sample (*N* CA+ = 638; *N* CA- = 501), using complete pairwise cases. As sensitivity analysis, we correlated the RF interrelations of the *full-information* networks with the RF interrelations of the *complete-information* networks (*N* CA+ = 508; *N* CA- = 443), which are based on listwise case deletion (which was applied in previous research, see for example Isvoranu et al., 2017). A high correlation would indicate that results are similar for both methods, and thus would support the soundness of *full-information* networks. For a detailed discussion of these results see Appendix B.8.

**Comparing CA+ and CA- networks.** To investigate the similarity of the CA+ and CA- network structures, we calculated the correlation of the RF interrelations of the two groups (i.e. CA+ and CA- network structure correlation). To examine the differences of the CA+ and CA- network structures, we applied four permutation tests (i.e. two-tailed; van Borkulo, 2018): Firstly, we tested whether the largest RF interrelation difference of the two networks (i.e. maximal edge weight difference) differs from the largest RF interrelation differences of randomly permuted network pairs, which functions as a network structure invariance test. Secondly, we tested whether the global network strength, i.e. the absolute sum of all RF interrelations, differs between the two network models (i.e. compared to permuted network model pairs). The global network strength indicates the overall network RF connectivity. Thirdly, we tested whether the global network expected influence (EI), i.e. the sum of all positive RF interrelations after subtracting the sum of the negative RF interrelations, differs between the two network models (i.e. compared to permuted network model pairs). The global network EI indicates the degree to which RFs enhance rather than hamper each other. Fourthly, we tested whether individual RF interrelations differ between the two networks (i.e. compared to the same individual RF interrelation differences between permuted network model pairs; please note, results for those tests without Holm-Bonferroni correction can be found in Appendix B.9; van Borkulo, 2018).

**Influence of the general distress variable on the RF networks.** To investigate the relationship between the RFs and an index that underpins mental health problems, we added the general distress variable to the networks. We then compared the resulting networks between the CA+ and CA- groups, by correlating the network structures between the two groups and by using the above

described permutation tests. Moreover, we examined whether potential differences between the CA+ and the CA- networks, upon taking the general distress variable into account, result from (1) differences in RF-RF interrelations, (2) differences in RF-distress interrelations, or (3) from both. To test whether group differences may result from changes in RF-RF interrelations, we tested whether RF-RF interrelations that are corrected for general distress levels (i.e. networks corrected for distress levels, but this time excluding RF-distress interrelations), differ significantly between the two groups. This comparison was again conducted through correlating the network structures of the two groups and through using the above described permutation tests. To test whether group differences may result from differences in RF-distress interrelations, we computed the Shortest Path Lengths ("shortest pathways") between the RFs and the general distress variable (i.e. the inverse of the absolute interrelation(s) between the respective RF and the general distress variable; Costantini et al., 2015; Isvoranu et al., 2017). The shortest pathway between two variables indicates the direct or indirect connection between those two variables along the strongest connection(s), or in other words the "quickest" way to traverse the network from the one variable to the other. Therefore, shortest pathways help to examine which RFs are mainly directly related to the general distress variable and which indirectly via other RFs. All network analyses were performed with the packages qgraph (Epskamp et al., 2012), mgm (Haslbeck & Waldorp, 2020), bootnet (Epskamp et al., 2018) and "NetworkComparisonTest" (NCT; van Borkulo, 2018), using R version 3.5.0 (R Core Team, 2017) in R Studio version 1.1.453.

**Data availability.** Data for this specific paper has been uploaded to the Cambridge Data Repository https://doi.org/10.17863/CAM.20806 and is password protected. Our participants did not give informed consent for their measures to be made publicly available, and it is possible that they could be identified from this data set. Access to the data supporting the analyses presented in this paper will be made available to researchers with a reasonable request to openNSPN@medschl.cam.ac.uk.

**Code availability.** Analysis code is available on https://osf.io/dm3fx/.

## 3.3 RESULTS

### 3.3.1 Sample

Neither gender nor age differed between the CA+ and the CA- group (see Table 3.1). Adolescents in the CA+ group had more often a psychiatric history, and on average a lower SES, and higher levels of depression and anxiety symptoms than adolescents in the CA- group.

### 3.3.2 RF and General Distress Comparisons between the CA+ and the CA- Group

The CA+ group (*n* = 638, 56% girls) reported lower levels for nine of the 10 RFs when compared to the CA- group (*n* = 501, 52% girls; see Table 3.2). Reflective rumination did not differ between the two groups. Furthermore, the CA+ group had higher general distress levels.

Table 3.1

*Sample comparisons: CA+ (n = 638) versus CA- (n = 501) groups*

| **Variable** | ***CA+*** | ***CA-*** | ***t*[*1]/*z*[*2]/*X*[2*3] (*DF*)** | ***p*** | ***95% CI*[*4]** |
|---|---|---|---|---|---|
| gender | *n* girls = 358<br>*n* boys = 280 | *n* girls = 262<br>*n* boys = 239 | 1.50(1) | .22 | |
| age | *M* = 14.49, *SD* = 0.28 | *M* = 14.48, *SD* = 0.28 | -0.43(1049.3) | .67 | -.04 - .03 |
| SES[*5] | *n* hard pressed = 77<br>*n* moderate means = 36<br>*n* comfortably off = 170<br>*n* urban prosperity = 37<br>*n* wealthy achievers = 318 | *n* hard pressed = 30<br>*n* moderate means = 11<br>*n* comfortably off = 105<br>*n* urban prosperity = 41<br>*n* wealthy achievers = 314 | 5.45 | <.001 | |
| psychiatric history (PH)[*6] | *n* PH = 201<br>*n* no-PH = 437 | *n* PH = 74<br>*n* no-PH = 427 | 42(1) | <.001 | |
| depression symptoms[*7] | *M* = 17.42, *SD* = 11.61 | *M* = 14.03, *SD* = 10.46 | -5.10(1088.5) | <.001 | -4.69 - -2.09 |
| anxiety symptoms[*8] | *M* = 16.92, *SD* = 12.61 | *M* = 13.92, *SD* = 11.28 | -4.17(1089.2) | <.001 | -4.42 - -1.59 |

*Note.* CA = childhood adversity. SES = socio-economic status. [*1]We applied Welsh's two-tailed independent sample t-test to account for potentially unequal variances across groups. [*2]As SES was split in five ordered categories, we applied the two-tailed Asymptotic Cochran-Armitage test (Hothorn, Hornik, van de Wiel, & Zeileis, 2008). [*3]We applied two-tailed Pearson's chi-square tests. [*4]The confidence interval (CI) for the difference in location estimates, corresponding to the alternative hypothesis. [*5]SES was assessed with the ACORN classification system (http://www.caci.co.uk; Morgan & Chinn, 1983). [*6]Psychiatric history was assessed with the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Present and Lifetime Version), additionally including learning disabilities, clinical sub-threshold diagnoses and deliberate self-harm (Kaufman et al., 1997). [*7]Depression symptoms were assessed with the Mood and Feeling Questionnaire (Messer et al., 1995). [*8]Anxiety symptoms were assessed with the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978).

Table 3.2

*RF and general distress comparisons: CA+ (n = 638) versus CA- (n = 501) groups*

| **Variable**[*1/*2] | ***CA+*** | ***CA-*** | ***t*[*3]/*X*[2*4] (*DF*)** | ***p*** | ***95% CI*[*5]** |
|---|---|---|---|---|---|
| Friendship support (high) | -0.07 | 0.06 | 2.23(1054.8) | .03 | .02 - .25 |
| Family support (high) | -0.08 | 0.09 | 2.79(1045.3) | .01 | .05 - .29 |
| Family cohesion (high) | -0.18 | 0.20 | 6.41(1066.4) | <.001 | .27 - .50 |
| Negative self-esteem (low) | -0.13 | 0.10 | 3.79(1071.5) | <.001 | .11 - .35 |
| Positive self-esteem (high) | -0.14 | 0.17 | 5.07(1070.9) | <.001 | .19 - .42 |
| Brooding (low) | -0.09 | 0.09 | 2.96(1046.4) | <.005 | .06 - .30 |
| Reflective rumination (low) | -0.06 | 0.01 | 1.21(1047.5) | .23 | -.05 - .19 |
| Distress tolerance (high) | -0.13 | 0.14 | 4.56(1072.4) | <.001 | .16 - .39 |
| Aggression (low) | low: 494 (score = 1)<br>high: 119 (score = 0) | low: 435 (score = 1)<br>high: 56 (score = 0) | 12.51(1) | <.001 | |
| Expressive suppression (low) | low: 408 (score = 1)<br>high: 209 (score = 0) | low: 366 (score = 1)<br>high: 129 (score = 0) | 7.56(1) | .01 | |
| General Distress | 0.13 | -0.16 | -4.85(1049.4) | <.001 | -.41 - -.17 |

*Note.* CA = childhood adversity. [*1]All RFs are scored in such a way that high values are protective (e.g. high levels of high friendship support or high levels of low negative self-esteem) and low values are harmful (e.g. low levels of high friendship support or low levels of low negative self-esteem). [*2]The continuous general distress variable is scored in such a way that the higher the value the higher the level of general distress. [*3]We applied Welsh's two-tailed independent sample t-test to account for potentially unequal variances across groups. [*4]We applied two-tailed Pearson's chi-square tests. [*5]The confidence interval (CI) for the difference in location estimates, corresponding to the alternative hypothesis.

### 3.3.3 Research Aim 1: RF Network Models

Firstly, we examined whether RFs are related to each other in both the CA+ and the CA- group networks. Both networks (see Figure 3.1.a.; or Appendix B.10) indicated positive relationships between most RFs. Three of the 45 RF interrelations differed in sign between the two groups. For example, low expressive suppression was associated with *low* friendship support in the CA+ network, but with *high* friendship support in the CA- network. A more detailed discussion of the interrelatedness of the RFs in the network models can be found in Appendix B.5. Robustness (see Appendix B.7) and sensitivity analyses (see Appendix B.8) indicated that the network models were stable and network parameters were estimated with a high accuracy.

Secondly, we investigated whether CA+ and CA- RF networks are dissimilar in structure. Contrary to our hypothesis, the CA+ and CA- group network structures were highly similar (i.e. correlation between the 45 regularized RF interrelations of each group; r = 0.94). Moreover, the network structure invariance test was not significant ($M$ = .17, permutations = 5000, $p$ = 0.21), and the CA+ and CA- networks did not differ with regard to the global network strength (i.e. the overall RF network connectivity; $S$ = .038, $S_{CA+}$ = 3.528, $S_{CA-}$ = 3.566, permutations = 5000, $p$ = 0.91). However, the networks did differ with regard to the global network expected influence (EI; $EI$ = .444, permutations = 5000, $p$ < .01). More specifically, the global network EI, which gives an indication of the degree to which RF are assumed to enhance each other, was significantly higher in the CA- compared to the CA+ group ($EI_{CA+}$ = 2.950, $EI_{CA-}$ = 3.394). We additionally compared all individual RF interrelations across the two networks, resulting in 45 Holm-Bonferroni corrected permutation tests: Only the RF interrelation between expressive suppression and friendship support differed significantly between the two networks ($E$ = 0.17, permutations = 5000, corrected $p$ < 0.01).

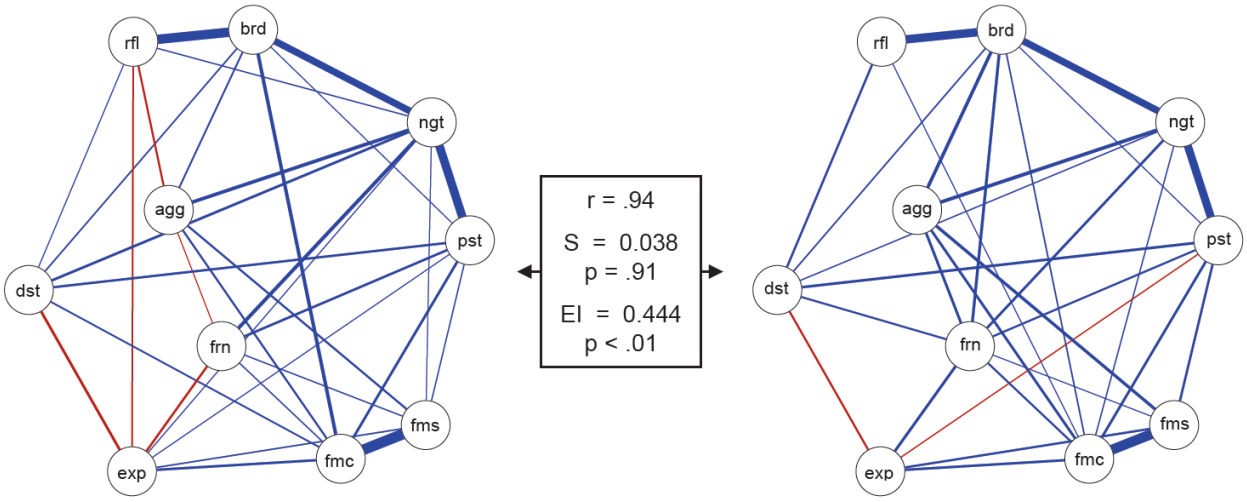
### 3.3.4 Research Aim 2: RF Network Models Including a General Distress Index

To explore the impact of general distress levels on the CA+ and CA- network structures, we next added the general distress variable to the networks (see Figure 3.1.b.). In the CA- group, all RFs were negatively related to general distress, except for expressive suppression and family support which were not related to general distress (Table 3.3.a.). In the CA+ group, all RFs were negatively related to general distress, except for expressive suppression which was positively related to general distress (shown in bold in Table 3.3.a.). Based on this unexpected finding for expressive suppression we performed further analyses (see Appendix B.12) which showed that most results remained similar when removing expressive suppression from the network models.

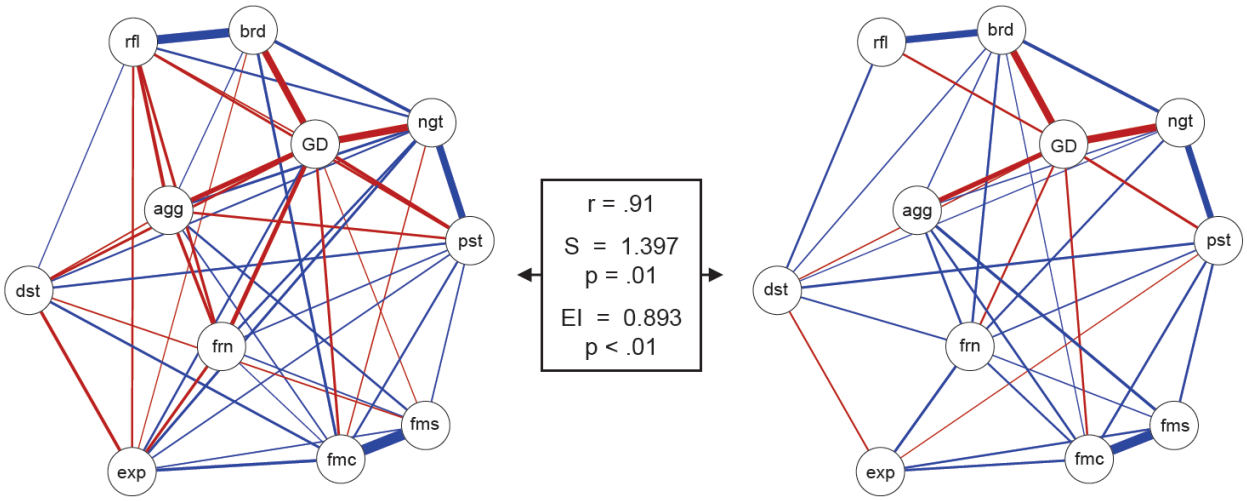
When adding the general distress variable to the networks, the CA+ and CA- network structures remained highly correlated (r = 0.91). Importantly however, the CA network structure invariance test was now significant ($M$ = .20, permutations = 5000, $p$ = 0.045), and networks also differed with regard to the global network strength ($S$ = 1.397, permutations = 5000, $p$ = 0.01), which was higher in the CA+ group ($S_{CA+}$ = 5.352, $S_{CA-}$ = 3.955). Along those lines, the global network EI was significantly lower in the CA+ compared to the CA- group ($EI$ = 0.893, permutations = 5000, $p$ < 0.01; $EI_{CA+}$ = 0.307, $EI_{CA-}$ = 1.200). For single interrelation comparisons we found, in line with the networks without the general distress variable, that only the interrelation between expressive suppression and friendship support

**CA+** **CA-**

1.a. RF Networks




1.b. RF Networks with the General Distress Variable




1.c. RF Networks Corrected for the General Distress Variable

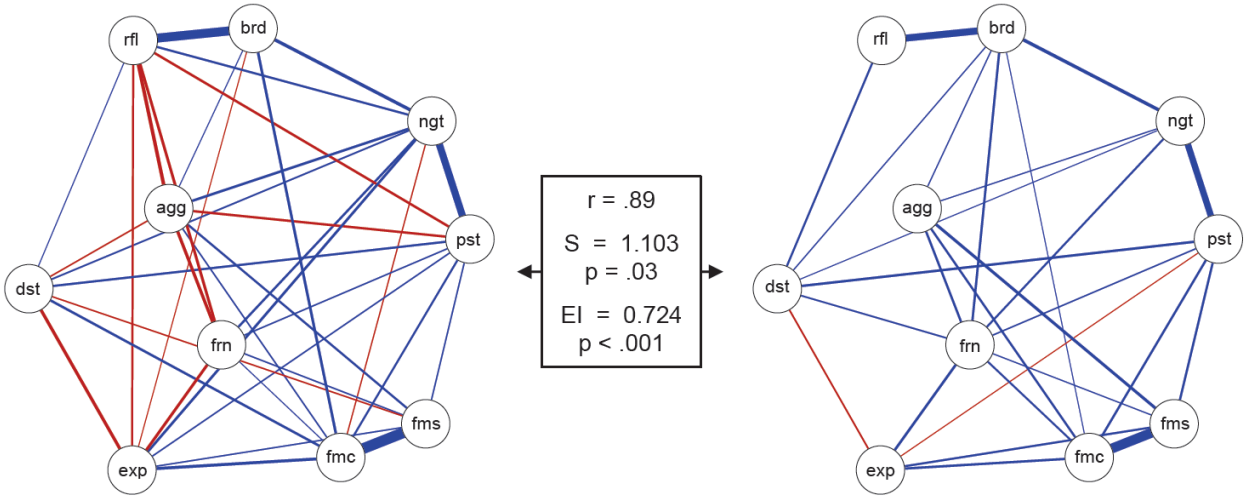


*Figure 3.1.* CA+ (n = 638) and CA- (n = 501) resilience factor networks without (1.a.), with (1.b.), and corrected for (1.c.) the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. Legend: Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the adjacency matrix correlation between the respective two networks (r), the difference in global network strength between the respective two networks (S), the difference in global network expected influence (EI) between the respective two networks (EI), and the p-value corresponding to the global network strength and global network EI comparisons (5000 comparison samples). The above networks with faded interrelations can be found in Appendix B.11.

differed significantly between the two networks ($E$ = 0.20, $N$ permutations = 5000, corrected $p < 0.001$). Ergo, upon adding the general distress variable to the networks, CA+ and CA- network structures differed not only with regard to the global network EI, but with regard to all examined structural measures. This finding may either be the result of (1) differing RF-RF interrelations between the two groups when taking general distress into account, (2) differing RF-distress interrelations between the two groups, or (3) of both. Therefore, we further examined those options.

### 3.3.5 Research Aim 3: Investigating Group Differences Due to the Influence of General Distress on the Network Models

**Group differences due to the influence of general distress on "RF-RF" interrelations.** Firstly, we explored whether differing CA+ and CA- network structures, after taking general distress into account, are the result of differing RF-RF interrelations between the two groups. To this end we compared the RF network structures that are corrected for the variance of the general distress variable, but do not include RF-distress interrelations, between the CA+ and CA- group. In other words, those networks contain only RF-RF interrelations that are corrected for general distress levels, but do not contain the general distress variable itself (see Figure 3.1.c.). The comparison of the resulting CA+ and CA- network structures revealed a correlation of .89. Moreover, we found that the network structure invariance test was significant ($M$ = .20, permutations = 5000, $p$ = 0.04), that the networks differed with regard to the global network strength ($S$ = 1.103, permutations = 5000, $p$ = 0.03), which was higher in the CA+ group ($S_{CA+}$ = 3.744, $S_{CA-}$ = 2.641), and also with regard to the global network EI ($EI$ = 0.724, permutations = 5000, $p < 0.001$), which was higher in the CA- group ($EI_{CA+}$ = 1.790, $EI_{CA-}$ = 2.514). For single interrelation comparisons we again found a significant difference for the interrelation between expressive suppression and friendship support ($E$ = 0.20, $N$ permutations = 5000, corrected $p < 0.01$). Hence, RF-RF interrelations differ significantly between the CA+ and CA- groups when correcting for general distress levels, both in terms of global network EI and global network strength (see Figure 3.1.c.). Along those lines Figure 3.1.c. shows that in the CA- network three RF-RF interrelations changed from positive to absent and all other interrelations kept the same relationship sign when being corrected for general distress levels. In contrast, in the CA+ network three RF-RF interrelations changed from positive to absent and seven interrelations changed from absent to negative. Moreover, the interrelatedness (or "centrality") coefficients of the RFs also changed slightly in both the CA+ and the CA- group, upon correcting for general distress levels (a discussion of those results can be found in Appendix B.6). Accordingly, differing CA+ and CA- network structures, when taking general distress

into account, are to some extent the result of general distress having a different impact on RF-RF interrelations in the two groups.

**Group differences regarding to "RF-distress" interrelations.** To scrutinize whether differing CA+ and CA- network structures, after taking general distress into account, are also the result of differing RF-distress interrelations between the two groups, we calculated the Shortest Path Lengths ("shortest pathways"; see Figure 3.2) between the RFs and general distress, and compared them between the groups. The shortest pathways indicate whether RFs have more direct or indirect connections with general distress (i.e. indirect connections go via intermediate RFs). Thus, a shortest pathway indicates the "quickest" way to traverse the network from the RF to the general distress variable. In the CA+ group, six RFs had a direct shortest pathway with general distress, whereas in the CA- group only three RFs had a direct shortest pathway. All other shortest pathways were indirect (for further details see Appendix B.13). This finding was particularly interesting, as the regularized partial correlations between the RFs and general distress appeared to be rather similar in the two groups (Pearson r = .92; Spearman r = .88; or see Table 3.3.a). Thus, the shortest pathways between RFs and general distress seemed for some RFs to differ between the two groups, despite the fact that the regularized partial correlations between the RFs and general distress were similar in the two groups. This may suggest that the differing RF-RF interrelations of the two groups facilitate more direct and less indirect RF-distress pathways in the CA+ compared to the CA- group. Accordingly, differing CA+ and CA- network structures, after taking general distress into account, seem to result from differing RF-RF interrelations, which in turn may lead to differing RF-distress pathways in the two groups.

Table 3.3
*Relationships between the RFs and the general distress variable*

**3.a. Regularized Partial Correlation Network**

| CA | negative SE | brooding | aggression | positive SE | reflection | family cohesion | friend support | distress tolerance | family support | expressive suppression |
|---|---|---|---|---|---|---|---|---|---|---|
| no | -0.40 | -0.37 | -0.24 | -0.10 | -0.07 | -0.07 | -0.06 | -0.02 | 0.00 | 0.00 |
| yes | -0.38 | -0.35 | -0.23 | -0.16 | -0.05 | -0.09 | -0.20 | -0.09 | -0.01 | **+0.06** |

**3.b. Association Network (i.e. Zero-Order Correlations)**

| CA | negative SE | brooding | positive SE | aggression | reflection | friend support | family cohesion | family support | distress tolerance | expressive suppression |
|---|---|---|---|---|---|---|---|---|---|---|
| no | -0.74 | -0.71 | -0.52 | -0.51 | -0.46 | -0.36 | -0.36 | -0.27 | -0.21 | -0.03 |
| yes | -0.75 | -0.71 | -0.57 | -0.36 | -0.45 | -0.37 | -0.43 | -0.33 | -0.31 | **+0.04** |

*Note.* CA = Childhood adversity (yes: *n* = 638, no: *n* = 501). SE = Self-esteem.

## 3.4 DISCUSSION

CA has deleterious consequences on adolescent mental health, and understanding how RFs facilitate good mental health is a fundamental goal of resilience research. Here we estimated RF network models for groups of adolescents with and without CA, in order to establish the first "over-arching theoretical construction" (p. 605 in Rutter, 1985) of how RF systems may facilitate mental health after CA. We found that the degree to which RFs enhance rather than hamper each other ("global network EI") was

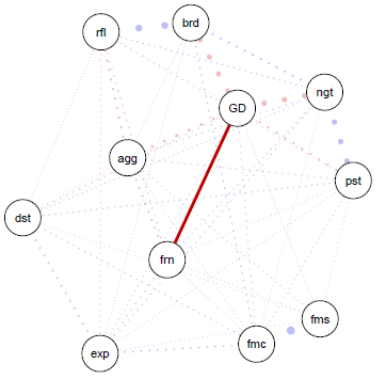
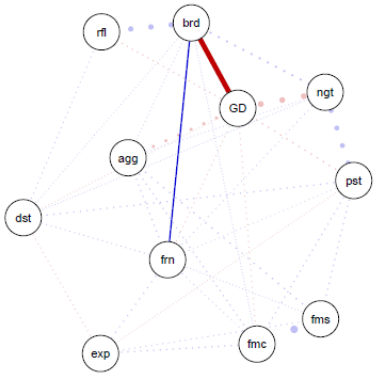
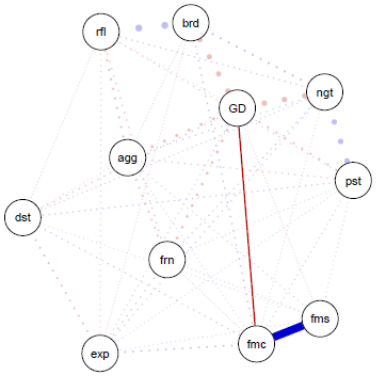
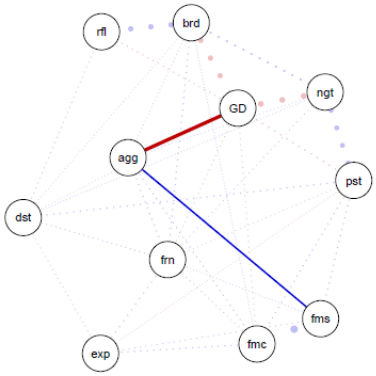
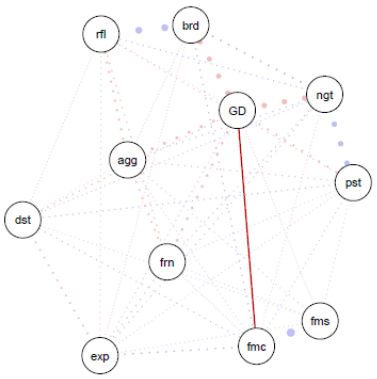
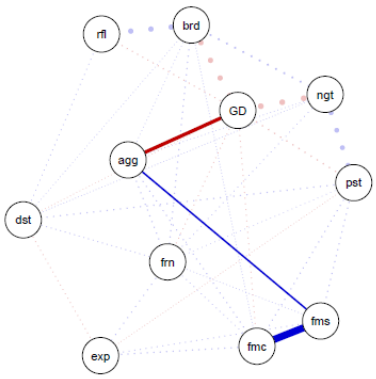
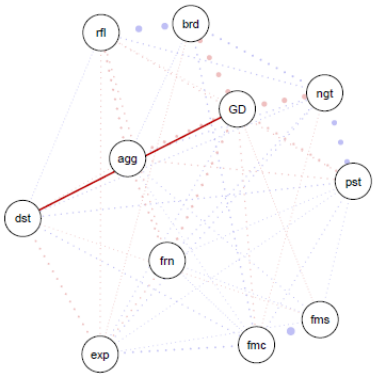
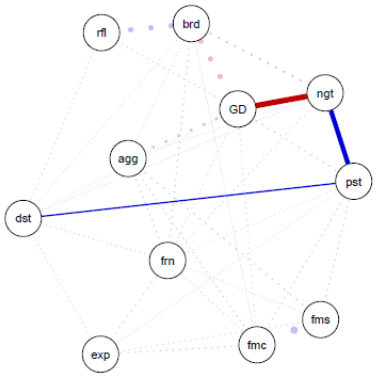
| CA+ | | CA- |
|---|---|---|
|  | Friendship support |  |
|  | Family support |  |
|  | Family cohesion |  |
|  | Distress tolerance |  |


*Figure 3.2.* Shortest pathways between the resilience factors (RFs) and the general distress variable, that differed between the CA+ (n = 638) and the CA- (n = 501) group. Non-transparent, continuous lines = shortest pathway of interest. Transparent, dotted lines = all remaining partial regularized correlation relationships. Positive interrelations = blue, negative interrelations = red. Legend: Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress.

significantly higher in the CA- compared to the CA+ group. Upon the correction of distress levels, RF-RF interrelations of the two groups additionally differed with regard to the overall network connectivity ("global network strength"). Moreover, interrelation pathways between RFs and concurrent general distress levels also seemed to differ between the two groups. Thus, differences between the CA+ and the CA- groups seem to be underpinned by both differences in how RFs relate with each other, as well as by differences in how RFs relate to general distress.

When we only investigated RF interrelations, without taking general distress levels into account, the overall RF network connectivity (or "global network strength") did not differ between adolescents with and without CA, and the maximal RF interrelation difference between the two groups (i.e. "network structure invariance test") was also not significant. We revealed those findings despite that the mean levels of nine out of ten RFs were higher in the CA- group than in the CA+ group. This may suggest that differing mean levels in RFs between groups do not necessarily lead to differences in the overall RF network connectivity between the groups. In a cross-sectional network model on depression symptoms, Schweren and colleagues (2018) did not detect a significant difference in the global network strength between strong- and weak treatment responders. Similarly, Snippe and colleagues (2017) applied dynamic network models on mental states prior to and after pharmacological as well as psychotherapeutic treatment for depression, and did not detect a global connectivity change in the pharmacological and only a marginal change in the psychotherapeutic treatment group, despite significant mean level reductions in depressive symptoms in both groups. Snippe and collegues (2017) tentatively concluded that the interrelations in the network models may represent an underlying "vulnerability" to depression rather than relate to mean-level changes in symptoms and mental states. Translated to our findings, this may indicate that the interrelated RF system may represent some underlying form of the group-level "resilient functioning capacity", regardless of mean-level differences in RFs between the two groups.

Interestingly, we found that in the CA+ network five RF interrelations had negative signs, which suggests that those RFs hamper rather than enhance each other. In the CA- network, however, only two interrelations had a negative sign, of which one was also negative in the CA+ network. When we compared the two network structures regarding the global network EI, which indicates the degree to which RFs enhance rather than hamper each other, we accordingly found a higher level of enhancement in the CA- compared to the CA+ group. This may be an indication for why adolescents exposed to CA have a higher liability of poor mental health (Greif Green et al., 2010).

Our findings further suggest that after taking distress levels into account, interrelations of emotional, behavioural, cognitive and social RFs not only result in a higher degree of RF enhancement in the CA- group, but also in a higher overall network connectivity in the CA+ group. More specifically, in the CA+ group, seven additional RF-RF interrelations were negative upon the correction of mental distress levels. Thus, whereas in the CA- network almost all RF interrelations are positive and thus may enhance each other, in the CA+ network more than a quarter of the RF interrelations are negative and thus may hamper each other. Negative RF-RF interrelations in the CA+ network, upon the correction for distress levels, may further underpin a deficient functioning of the RFs, and thus may also be a reason for why adolescents with exposure to CA are on average more vulnerable for subsequent distress.

Our findings additionally showed that the CA+ group had lower levels of RFs and higher levels of general distress. Therefore, a higher vulnerability to distress in the CA+ group may be substantiated by (1) high distress leading to lower RFs, (2) lower RFs leading to higher distress, or (3) by unfavourable, mutualistic RF-distress associations (e.g. mutualistic coupling; Kievit et al., 2017). Moreover, the CA+ group had more direct connections between RFs and concurrent general distress, compared to the CA- group. More specifically, in the CA- group only three RFs had direct shortest pathways with general distress, whereas in the CA+ network six RFs had direct shortest pathways with general distress. In case of *high* RFs and *low* general distress, many direct RF-distress pathways may be advantageous, as many high RFs then directly can contribute to lower distress levels (and/or vice versa). Yet, in case of *low* RFs and *high* distress, as in the CA+ group, many direct RF-distress pathways may be disadvantageous, as high distress then directly can contribute to many low RFs (and/or vice versa). Hence, lower levels of RFs and higher levels of distress, together with more direct RF-distress relationships, may be another reason for why adolescents with exposure to CA are on average less protected from subsequent distress.

Given our finding for the CA- group, that almost all RFs were positively interrelated, it is likely that enhancing the most strongly connected RFs in the RF system may spread through the network and thereby enhance the level of other RFs. Many positively interrelated RFs that enhance each other, may in turn lower concurrent, and thus potentially also subsequent mental distress. In contrast, in the CA+ network many RF interrelations were negative. Therefore, enhancing RFs in the CA+ network may not be sufficient to effectively reduce distress levels, as higher levels of RFs may even further hamper other RFs. However, reducing general distress levels in the CA+ network could perhaps be achieved by intervening on negative RF-RF interrelations to turn them into positive interrelations, so that RFs enhance rather than hamper each other. Examining this should be subject of future research.

A potentially important negative RF-RF interrelation in the CA+ network is the “expressive suppression – friendship” interrelation, which differed significantly from the corresponding positive RF-RF interrelation in the CA- network. This finding suggests that, in the CA+ group, (1) ineffectively communicating emotions drives friendship withdrawal, (2) friendship withdrawal drives ineffectively communicating emotions, or (3) both drive each other reciprocally (reciprocal coupling). For example, it may be that CA exposure results in higher manifest levels of negative emotions (Dunn et al., 2011); and showing these emotions may burden friendships and/or reduce socializing behaviours in peers. Alternatively, an already existing low level of friendships and socializing may generate more negative emotions and thus support an increased expression of those emotions. Translational research could test whether training CA-exposed adolescents to communicate their own emotions better, may lead to improved friendships. If our finding of potentially dysfunctional RF interrelations in the CA+ group holds up in replication over time and in independent samples, this may explain why individuals with a history of CA are on average less likely to respond to treatment for mental health problems than individuals without a history of CA (Nanni, Uher, & Danese, 2012).

Our study is not without practical limitations. First, CA was assessed retrospectively, which has the disadvantage of potential recall bias (Goodyer et al., 2010). Second, CA was classified as a binary variable, categorizing “any” versus “no” history of CA. Such a categorization is rather crude, as it assumes that any form of adversity, irrespective of the severity and frequency, contributes to a difference in mental health between CA+ and CA- groups. Instead, the effects of CA on the general distress variable may be linear (the more CA, the higher the probability of general distress) or U-shaped (e.g. challenge or inoculation theory; no or high CA goes together with high general distress, moderate CA goes together with low general distress; Fergus & Zimmerman, 2005; Masten, 2011; Zolkoski & Bullock, 2012). However, prior analysis of our adversity data demonstrated that, in our sample, CA could not be modelled as a single continuous variable, as a one factor CFA model did not fit the data (Dunn et al., 2011). Moreover, clustering CA in multiple classes would, for the analyses presented here, not have been possible in terms of power. Third, we mainly measured family-related adversities, which may limit the generalization to other types of CAs such as peer to peer bullying. Fourth, only a subset of empirically supported RFs (see Chapter 2) was measured in ROOTS. The restricted number of included RFs may impact the network structure and may limit the content validity. Fifth, all RFs were solely assessed after the exposure to CA. Therefore, the study design does not allow for the establishment of baseline RF interrelations prior to CA. Ergo, we cannot draw conclusion with regard to the extent to which RFs change from pre to post CA (Bonanno et al., 2015; Kalisch et al., 2015). Sixth, some variables had missing data. This led in the complete-information samples to 11.58 percent less data for the CA- group and 20.38 percent less data for the CA+ group, when compared to the respective full-information samples for CA+ and CA- groups. Seventh, our data modelling procedure was conducted in two steps: (a) deriving RF scores from categorical CFAs and (b) estimating network models for the resulting RFs. Future studies may want to look into latent network modelling, which is a novel methodology that efficiently performs both steps at one time (Epskamp, Rhemtulla, & Borsboom, 2017).

Our study also contained theoretical limitations. First, as all estimated networks were cross-sectional, the general distress variable was assessed at the same time as the RFs. Hence, it is likely that the psychological state of the adolescents influenced their self-ratings (and parent-ratings) of RFs. Therefore, the network models with the general distress variable mainly serve as a proof of principle, to check that the RFs are indeed related to general distress. It is important that future studies investigate the predictive values of the RFs, through scrutinizing the interrelations between RFs and subsequent general distress. Second, and relatedly, as all our measures were assessed cross-sectionally as well as via the same method (i.e. questionnaires), there may be a risk of "consistency motifs, implicit theories, social desirability tendencies, dispositional and transient mood states, and any tendencies on the part of the rater to acquiesce or respond in a lenient manner" which can induce common methods bias (p. 887 in Podsakoff, MacKenzie, Lee, & Podsakoff, 2003; who provide a detailed discussion of this bias). Common methods bias "can inflate, deflate, or have no effect on the observed relationships" (p. 893 in Podsakoff et al., 2003). However, the risk of common methods bias is likely to have been limited in our results, as (a) various RFs differed in response format (i.e. content of response options and number of response options), and (b) two RFs were not assessed via adolescent but via caregiver report (i.e. distress tolerance and expressive suppression) and still fitted well into the network models, both of which at the least reduces common methods variance. Third, as our expressive suppression factor was assessed with only one item, our expressive suppression factor may have lacked specific aspects of the concept (i.e. content validity), or we may have measured a different construct than prior research (i.e. construct validity). Tapping a potentially different aspect of expressive suppression may explain our contrasting findings with the literature (Boyes et al., 2015; Gross & John, 2003; i.e. we found that in the CA+ group the expressive suppression RF had a positive relationship with the general distress variable) and requires clarification in future studies. Yet, removing expressive suppression from the network models did only slightly alter our findings. Fourth, it is interesting to note that, in the CA+ network, some RF-RF interrelations are negative upon controlling for the general distress variable. The explanation we put forward for this finding is that the result is due to different network structures in the CA+ and CA- groups. Alternative statistical explanations for this result exist, such as conditioning on a collider. Conditioning on a collider (in this case general distress) can induce spurious negative relationships among variables (Elwert & Winship, 2014), similar to what we observed in the CA+ network once entering general distress. However, given that this only occurred in the CA+ network, despite rather similar RF-distress (regularized partial) correlations in the two groups, conditioning on a collider does not plausibly seem to be the main explanation for the negative RF interrelations, as one would expect this to happen in an equal manner in the CA- network (for further discussion see Appendix B.14). In our sample eight of the 10 RFs functioned as mediators (indirect effects) and one additionally as moderator (interaction effect) for the relationship between CA and general distress, which may perhaps help explain why the correction for distress levels had differing effects on the RFs in the CA+ compared to the CA- group (see Appendix B.14). Fifth, it is crucial to note that our findings are derived from group level analyses, and thus represent averages across all participants. Therefore, our findings may not directly translate to person specific levels and thus may not apply to all adolescents. For clinical purposes, RF interrelations should be evaluated on an individual level.

Besides those limitations, our study also has notable strengths. For example, our study combines several advanced statistical methods - i.e. categorical CFAs, latent class analysis, bifactor modelling, and network analysis - and thereby accomplished to be the first study to model a complex system of RFs. Moreover, as all included RFs were empirically found to moderate and/or mediate the positive relationship between CA and mental health problems (see Chapter 2), I believe that our RF models represent the construct we intended to measure well and thus achieved high construct validity.

To the best of my knowledge, this is the first time that network analysis has been applied to establish the interrelatedness of empirically supported RFs. I draw several conclusions aimed at aiding the refinement of resilience theory as well as the development of translational research regarding mental health resilience following CA. Yet, our findings require replication across time and in independent samples. Our findings suggest that the degree to which RFs enhance rather than hamper each other ("global network EI") was significantly higher in the CA- compared to the CA+ group. Moreover, upon correction for general distress levels, the RF networks additionally differed with regard to the global RF connectivity. More specifically, in the CA- network almost all RFs were positively interrelated and thus may enhance each other, whereas in the CA+ network some RFs were negatively interrelated and thus may hamper each other. Moreover, the CA+ group showed more direct relations between RFs and the general distress variable. Thus, differences between the CA+ and the CA- groups seem to be underpinned by both differences in how RFs relate with each other, as well as by differences in pathways between RFs and general distress. Translational research could explore whether intervening on negative RF-RF interrelation, so that they turn positive and RFs can enhance each other, may alter RF-distress relations, resulting in a lower risk for subsequent mental health problems.

Building on the here established knowledge, in the next chapter, I take the question of how RFs are interconnected a step further and will try to unravel changes of the potentially protective RF system between early (age 14) and later adolescence (age 17).

# CHAPTER 4

# Unravelling the Complex Nature of Resilience Factors and their Changes between Early and Later Adolescence

Adolescents who have been exposed to adversity in childhood (CA), such as traumatic and/or severely stressful events, have a higher risk of developing mental health problems (Greif Green et al., 2010; Kessler et al., 2010; McLaughlin, 2016). As introduced in the previous Chapters, approximately one in two children and adolescents worldwide experience adverse events before the age of 18 (Greif Green et al., 2010; Kessler et al., 1997, 2010; McLaughlin, 2016). Therefore, it is imperative that the deleterious mental health consequences following CA are addressed in research, therapy and mental health policy. This notion has not only been noticed in science (Afifi et al., 2016; McLaughlin, 2016), but has also led to a discussion in public media questioning whether "childhood trauma [should] be treated as a public health crisis?" (Blakemore, 2018, in the National Public Radio, 09 November 2018) and whether "people [can] be saved from a terrible childhood?" (Zanolli, 2018, in The Guardian, 07 November 2018). One way to understand better how we can reduce the deleterious consequences of CA is to study the complex nature of resilience factors (RFs); i.e. factors that are empirically found to reduce the risk of mental health problems following CA (see Chapter 2 or Fritz, de Graaff, et al., 2018; Zimmerman et al., 2013). To this end, I here aim to shed light on the longitudinal nature of RFs between two time points, respectively marking early and later adolescence.

RFs operate on various intertwined functioning levels encompassing biological (e.g. genes or hormones), intra-personal (e.g. distress tolerance), and inter-personal levels (e.g. peer support; see Chapter 2; Ioannidis, Askelund, Kievit, & van Harmelen, 2020; van Harmelen et al., 2016). As in the other Chapters, I will focus on the latter two categories as those RFs can be targeted in psychosocial interventions and may therefore be particularly relevant in informing translational research and thus eventually prevention and therapy.

Despite the fact that RFs do not function in isolation, most studies have investigated single RFs (e.g. Chapter 2; Diehl et al., 2012). Recently, researchers have argued that to improve our understanding of resilience mechanisms, it is necessary to move from relatively simple reductionist towards more holistic, complex models (Diehl et al., 2012; Kalisch et al., 2019; Scheffer et al., 2018). In several research fields, complex system models have been applied to describe risk and resilience processes, as for instance for financial markets or ecosystems (Battiston et al., 2016; Scheffer et al., 2015, 2018). Complex system models promise to fit the complexity of resilience research well, as they enable the exploration of multiple interconnected factors that are assumed to reinforce each other. Recently, my colleagues and I took the first step in bridging this gap for resilience research, focussing on mental health in the face of adversity (see Chapter 3 or Fritz, Fried, Goodyer, Wilkinson, & van Harmelen, 2018). We showed that RFs function as a complex interrelated network in both adolescents with and without CA, at age 14. We found that the group of adolescents with CA had lower RF mean levels and the RFs were less positively interrelated, suggesting that the RFs may not enhance each other to the same extent as in adolescents without CA.

Mental health levels can change over time, particularly during the process of dealing with adversity (Costello, Copeland, & Angold, 2011; Kalisch et al., 2017; Masten, 2011; Rutter, 1985). This suggests that RFs and/or their interrelations may also change over time. Individuals with CA often have lower levels of RFs (e.g. Chapter 2; Almquist et al., 2018), which are suggested to be transferred forward across development (Kim, Oesterle, Catalano, & Hawkins, 2015; McLaughlin, 2016). Hence, it is crucial to determine how RFs change over time in adolescents with and without CA, as this firstly unravels whether RFs change similarly or differently in the two groups; and secondly reveals which RFs improve, deteriorate or stay stable during adolescence. Such RF changing patterns can inform translational research which in turn can shed light on the RFs that should be targeted and promoted to aid successful development after CA (Kim et al., 2015; McLaughlin, 2016). However, research on RF changes is surprisingly scarce, and results are mixed: Some intra- and inter-personal RFs are found to increase (e.g. ruminative worrying, prosocial involvement), whereas others have been reported to stay stable between early and later adolescence (e.g. family involvement, expressive suppression, dysfunctional rumination; Frydenberg & Lewis, 2000; Kim et al., 2015; Zimmermann & Iwanski, 2014). Here, my colleagues and I shall therefore examine whether RFs change between early (age 14) and later (age 17) adolescence, through investigating (a) RF mean levels, (b) RF interrelations, and (c) the way RFs are interrelated with distress (directly and/or indirectly via other RFs). Importantly, we specifically examine whether RFs change differentially in groups of adolescents with (CA+) and without CA (CA-).

## 4.2 METHODS

### 4.2.1 Design

In 2005 and 2006, 1238 14-year-old adolescents were recruited from schools in Cambridgeshire to take part in the longitudinal ROOTS study. Follow-up took place around age 17 (Goodyer et al., 2010). Consent was provided by the adolescents and one parent (Goodyer et al., 2010). ROOTS was conducted following Good Clinical Practice guidelines and the Declaration of Helsinki, and was approved by the Cambridgeshire Research Ethics Committee (03/302; Dunn et al., 2011).

### 4.2.2 Sample

In the current study we performed all main analyses on 1130 of the 1238 participants. We included all those who had data for potential CA experiences (CA+: n = 638; CA-: n = 501) and had less than 85% missingness on the analyses variables (*n* = 1188), resulting in 631 adolescents with and 499 adolescents without prior exposure to CA.

### 4.2.3 Childhood Adversity (CA)

CA was assessed with the semi-structured Cambridge Early Experience Interview (CAMEEI) that mainly measures intra-family related adversity before the age of 14 (Dunn et al., 2011). The interview was conducted with the primary caregiver, which was in 96% of the cases the biological mother. All interviews were performed when the adolescents were 14 years old. The CAMEEI was designed to

measure adverse events in three time windows (0-5, 5-11, and 11-14 years), to support recall accuracy. Several types of adverse experiences were measured: loss of a family member, family separations (> 6 months), divorce, death, adoption, discord within the family, absence of maternal affection/involvement, aberrant parenting style, significant medical illnesses within the family, psychopathology of family members, times of parental unemployment, financial hardship, physical abuse, sexual abuse, emotional abuse, criminality of family members, acute life events (e.g. environmental event with impact on the living situation), and chronic social hardship (e.g. demands of caring for extended family; Dunn et al., 2011). Based on this information Dunn and colleagues (2011) performed a latent class analysis, which revealed four classes (no CA, moderate CA, severe CA, and aberrant parenting CA) for each of the three time windows. In line with previous reports (see e.g. Chapter 3) adolescents were assigned a "0" when they belonged for all three time windows to the "no CA" category (CA-), and were assigned a "1" when they belonged for at least one time window to a category other than "no CA" (CA+; see Table 4.1 for detailed numbers).

Table 4.1
*Numbers CA exposure (CA+ = 638, CA- = 501)*

| **0 to 5 years** | **5 to 11 years** | **11 to 14 years** | **CA variable** | **Cumulative number of participants with CA** | | |
|---|---|---|---|---|---|---|
| CA+ = 355 | CA+ = 463 | CA+ = 406 | CA+ = 638 | 1 time window | 2 time windows | 3 time windows |
| CA- = 784 | CA- = 676 | CA- = 733 | CA- = 501 | n = 262 | n = 166 | n = 210 |

*Note.* CA = childhood adversity.

### 4.2.4 General Distress

To compile a general distress index, we used the 13-item short form of the Mood and Feelings Questionnaire (MFQ; Messer et al., 1995), measuring a broad range of depression related symptoms, and the 28-item Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978) measuring a wide range of anxiety related symptoms. We used confirmatory factor analysis (CFA) based on polychoric correlations to estimate one underlying latent general distress factor for those 41 items. Brodbeck et al. (2011), Stochl et al. (2015) and St Clair et al. (2017) used similar approaches and showed that a latent general distress factor replicates well in adolescent samples. Please note, for computational reasons we have used fewer depression items for the general distress factor than in Chapter 3 (for a detailed rationale see Appendix C.1).

### 4.2.5 Resilience Factors (RFs)

Based on findings of our preregistered systematic review (see Chapter 2), we included 8 self-report (1-8 below) and 2 parent report RFs (9-10 below) that were assessed in our adolescent cohort. All RFs are scored in such a way that high values are protective, to which end five of the scales were reversed:

1. Friendship support was assessed with five items of the Cambridge Friendships Questionnaire (Goodyer et al., 1989).
2. Family support was assessed with five items of the McMaster Family Assessment Device (Epstein et al., 1983).

3. Family cohesion was assessed with seven items of the McMaster Family Assessment Device (Epstein et al., 1983).
4. Positive self-esteem was assessed with five items of the Rosenberg self-esteem scale (Rosenberg, 1965).
5. Negative self-esteem was assessed with five items of the Rosenberg self-esteem scale (Rosenberg, 1965). We reversed the items so that high values of low negative self-esteem are protective.
6. Reflective rumination was assessed with five items of the Ruminative Response Scale (RRS; Burwell & Shirk, 2007; Treynor et al., 2003). We reversed the items so that high values of low reflective rumination are protective.
7. Ruminative brooding was assessed with five items of the RRS (Burwell & Shirk, 2007; Treynor et al., 2003). Please note the ruminative brooding factor does not match the one used in Chapter 3, for a detailed rationale see Appendix C.1 and C.2. We reversed the items so that high values of low ruminative brooding are protective.
8. Aggression was assessed with four items of the Behaviour Checklist (11 questions based on the DSM-IV criteria for conduct problems; American Psychiatric Association, 2000; Goodyer et al., 2011). We reversed the items so that high values of low aggression are protective.
9. Distress tolerance was assessed with five items of the Emotionality Activity Sociability Temperament Survey (Bould et al., 2013).
10. Expressive suppression was assessed with one item of the Antisocial Process Screening Device (Poythress et al., 2006). We reversed the item so that high values of low expressive suppression are protective.

More detailed information regarding the exact content of the RF constructs can be found in Chapter 3. Information regarding the psychometric properties of the RF measures is reported in Appendix B.2.

### 4.2.6 Analysis

All analyses were conducted with R version 3.5.1 (R Core Team, 2018). All used packages and the belonging version numbers can be found in Appendix C.3.

**Variable preparation.** A minor subset of participants had incidentally missing items and some participants had missingness due to attrition, both detailed in Appendix C.4 Table C.2. The identified missingness patterns on most RFs and general distress could partially be accounted for by exposure to CA, being male, having a low mood, and having a psychiatric history prior to the age of 14 (see Appendix C.4 Table C.3). Accordingly, we used multivariate multiple imputation algorithms with chained equations to impute the missing data (van Buuren & Groothuis-Oudshoorn, 2011). We computed 10 imputation data sets each with 100 iterations, using predictive mean matching algorithms for ordered categorical items and logistic regression for dichotomous items. The imputation models were based on seven descriptive variables (CA, gender, socio-economic status, prior psychiatric history at occasion 1 and 2, and age at occasion 1 and 2), as well as 50 RF, 33 depression-related, and 28 anxiety-related items for both occasions, resulting in a total of 229 items. In contrast to missingness on the RF or distress variables, we did not impute data for the CA variable. We made this decision as we felt that

some forms of CA, such as a traumatizing car crash or being exposed to fire in the home, are not sufficiently predictable to be imputed for missingness. The imputed data sets contained data for 1188 participants. To estimate the best fitting latent RF and distress indices we used CFA models and extracted the resulting factor scores as RF and general distress variables. We decided to use factor scores instead of sum scores to reduce measurement error and to circumvent tau-equivalence (for a rational see Appendix C.5 Part A). As we aimed to compare two time points, we estimated longitudinal CFAs (LCFAs; separately for each RF and general distress). Given that all RF and general distress items were assessed with three to six answer categories, we computed categorical LCFAs (Wu & Estabrook, 2016), treated the items as ordinal and used a weighted least square mean and variance adjusted (WLSMV) estimator (for details see Appendix C.5 Part B). Distribution plots for the RFs and general distress are in Appendix C.5 Figure C.5. Hence, all main analyses were performed on 1130 participants (CA+ $n$ = 631, CA- $n$ = 499) who had data for potential CA experiences ($n$ = 1139) and had less than 85% missingness on the analyses variables ($n$ = 1188). In contrast to the analyses, all descriptive statistics are computed on the un-imputed data and may therefore contain slightly different sample sizes. The interested reader can find analysis results not being based on imputed data in online Supplement I.

**Investigating RF mean level changes.** To examine whether RFs (a) differ in their protective value between the CA+ and the CA- group, and (b) change in their protective value between age 14 and 17, we conducted RF mean comparison analyses. More specifically, we compared the RF and general distress mean levels (a) between the CA+ and the CA- group (i.e. separately for age 14 and 17), and (b) between age 14 and age 17 (i.e. separately in the CA+ and CA- groups). To ensure latent mean comparability across ages we estimated strongly invariant categorical LCFAs (Wu & Estabrook, 2016), for which the exact LCFA parameter specifications and model identification details are outlined in Appendix C.5 Part B. All strongly invariant categorical LCFAs fitted satisfactorily (Appendix C.5 Part B, Table C.5). We did not compute an LCFA for the expressive suppression RF, as this RF was measured with only one item. We binarized the aggression and expressive suppression RFs, as they showed a restricted range. To circumvent slight deviations from normality we tested CA+ vs CA- mean level differences with independent sample Wilcoxon Rank Sum tests (with continuity correction). Moreover, we compared age 14 and age 17 mean levels with paired sample Wilcoxon Signed Rank tests (with continuity correction). As sensitivity analyses, we re-ran the mean change analyses (a) with factor scores retrieved from the full invariance models (see Appendix C.6), and (b) with sum scores (see Appendix C.6). All mean comparisons were corrected for the false discovery rate (Benjamini & Hochberg, 1995). Additionally, we explored whether CA moderates the relationship between age and RFs, to test whether the change patterns of the RFs differ between the two groups.

**Investigating network structure changes.** To examine (a) whether RFs interrelate differently in the CA+ and the CA- groups, and (b) whether those RF interrelations change between age 14 and 17, we computed RF network models. More specifically, we used RF factor scores to estimate regularized partial correlation network models (Epskamp & Fried, 2018). Those models were computed separately for adolescents with and without CA, as well as for age 14 and age 17. We compared the resulting models with each other using permutation tests (van Borkulo, 2018). To ensure that the

exchangeability assumption of permutation tests was met (i.e. the joint distribution of the scores is invariant when permuting over time), we estimated fully invariant categorical LCFAs. The exact LCFA parameter specifications and details regarding the model identification can be found in Appendix C.5 Part B. All fully invariant categorical LCFAs fitted satisfactorily (see Appendix C.5 Part B Table C.5). As above, we did not compute an LCFA for expressive suppression, and we again binarized the aggression and expressive suppression RFs. We estimated (a) networks only containing the 10 RFs, (b) networks containing both the 10 RFs and the general distress factor, and (c) networks containing the 10 RFs corrected for general distress levels. To ensure conciseness, I here discuss the RF network models being corrected for general distress levels, as those enable the comparison of the CA+ and the CA- groups when taking the putatively confounding effect of psychopathology levels into account. The other two models are discussed in Appendix C.7.

For the comparisons of the four network models (i.e. CA+ vs CA- = independent sample permutation tests; and age 14 vs age 17 = paired sample permutation tests) we conducted three types of network comparison tests (van Borkulo, 2018). Firstly, we investigated whether the highest interrelation difference between the respective two networks differs from the highest interrelation differences of several (i.e. 5000 permutations) randomly permuted network model pairs, which indicates whether the two tested network structures are invariant (van Borkulo, 2018). Secondly, we investigated whether the relative connectivity, which is the sum of the positive interrelations after subtracting the sum of the negative interrelations, differed between the two respective networks. This test is also called "global network expected influence" comparison (e.g. Chapter 3; Elliott, Jones, & Schmidt, 2020) and indicates to which degree RFs are concurrently positively associated. This test is of particular interest here, as it suggests to which degree RFs can concurrently enhance each other. Thirdly, we explored which individual RF interrelations and/or interrelations between RFs and general distress differed between the respective two networks of interest (van Borkulo, 2018). Hence, the first two tests examine *global* network structure differences, whereas the third test examines *local* network structure differences.

**Investigating "RF-distress" pathway changes.** To examine the way RFs are interrelated with distress in the network models, we calculated two types of pathways between the RFs and general distress. First, we examined the direct pathways between the RFs and general distress, regardless of whether those pathways are the strongest or "quickest" ways to traverse the network from the RFs to general distress (Isvoranu et al., 2019). Second, we examined the shortest pathways (or "shortest path lengths") between the RFs and general distress, regardless of whether the RFs have direct pathways with general distress. More specifically, we explored whether the shortest pathway to traverse the network from a given RF to the general distress variable is direct, or indirect via other RFs (Isvoranu, Borsboom, Van Os, & Guloksuz, 2016). Moreover, we conducted permutation tests to compare the two types of pathways between the CA+ and the CA- group, for both age 14 and age 17. Lastly, we examined whether the two types of pathways changed between age 14 and 17 (i.e. separately for the CA+ and the CA- groups), again using permutation tests. Correlations and regularized partial correlations between the RFs and the general distress variable, for both CA+ and CA- as well as for age 14 and age 17, are discussed in Appendix C.8.

**Network stability, accuracy, and inference.** To test the robustness of our network model parameters we estimated the stability of expected influence (EI) coefficients and the accuracy of all interrelations. We tested the stability of the EI coefficients by applying a subset bootstrap (2000 bootstraps) to identify the maximum sample percentage that can be dropped to reveal (with a 95% chance) a relationship of ≥0.7 between the subset and the original EI coefficients (Epskamp et al., 2018). Moreover, we tested the accuracy of the network models by bootstrapping all interrelations (2000 bootstraps) and investigating their bootstrapped confidence intervals (Epskamp et al., 2018). Those analyses are reported in Appendix C.9. We further explored the node expected influence coefficients for individual RFs (i.e. the sum of all positive interrelations of the respective RF, after subtracting the sum of the negative interrelations of that RF; Costantini et al., 2015; McNally, 2016), which are reported in Appendix C.10.

**Network sensitivity analyses.** To establish whether our results would hold if the RFs were computed differently, we re-estimated the network models (a) based on factor scores of the configural LCFAs, which do not constrain parameters across time points but estimate the best fitting time point specific latent factor, and (b) based on sum scores. Results were overall similar and are discussed in Appendix C.11 and C.12.

**Data availability.** Data for this specific paper has been uploaded to the Cambridge Data Repository https://doi.org/10.17863/CAM.36708 and is password protected. Our participants did not give informed consent for their measures to be made publicly available, and it is possible that they could be identified from this data set. Access to the data supporting the analyses presented in this paper will be made available to researchers with a reasonable request to openNSPN@medschl.cam.ac.uk.

**Code availability.** Analysis code is available on https://osf.io/mwx2t/.

## 4.3 RESULTS

### 4.3.1 Sample

The CA+ and the CA- groups did not differ with regard to age or gender, but the CA+ group had a lower socio-economic status (see Table 4.2). In addition, adolescents in the CA+ group were more likely to have a psychiatric history, and had higher levels of depression and anxiety symptoms, at both age 14 and 17.

Table 4.2
*Sample comparisons: CA+ (n = 638) versus CA- (n = 501) groups*

| | CA+ | CA- | $t^{*1}/z^{*2}/X^{2*3}$ (*DF*) | 95% CI[*4] | *p* |
|---|---|---|---|---|---|
| gender | *n* girls = 358<br>*n* boys = 280 | *n* girls = 262<br>*n* boys = 239 | 1.50(1) | | .22 |
| SES[*5] | *n* hard pressed = 77<br>*n* moderate means = 36<br>*n* comfortably off = 170<br>*n* urban prosperity = 37<br>*n* wealthy achievers = 318 | *n* hard pressed = 30<br>*n* moderate means = 11<br>*n* comfortably off = 105<br>*n* urban prosperity = 41<br>*n* wealthy achievers = 314 | 5.45 | | <.001 |
| **Age 14** | | | | | |
| age | *M* = 14.49, *SD* = 0.28 | *M* = 14.48, *SD* = 0.28 | -0.43(1049.3) | -.04 - .03 | .67 |
| psychiatric history (PH)[*6] | *n* PH = 201<br>*n* no-PH = 437 | *n* PH = 74<br>*n* no-PH = 427 | 42(1) | | <.001 |

| | | | | | |
|---|---|---|---|---|---|
| depression symptoms | *M* = 17.42, *SD* = 11.61 | *M* = 14.03, *SD* = 10.46 | -5.10(1088.5) | -4.69 - -2.09 | <.001 |
| anxiety symptoms | *M* = 16.92, *SD* = 12.61 | *M* = 13.92, *SD* = 11.28 | -4.17(1089.2) | -4.42 - -1.59 | <.001 |
| **Age 17** | | | | | |
| age | *M* = 17.49, *SD* = 0.34 | *M* = 17.48, *SD* = 0.32 | -0.56(1017.5) | -.05 - .03 | .58 |
| PH[*6] | *n* PH = 268<br>*n* no-PH = 297 | *n* PH = 122<br>*n* no-PH = 345 | 48.48(1) | | <.001 |
| depression symptoms | *M* = 16.36, *SD* = 12.27 | *M* = 12.38, *SD* = 10.19 | -5.51(967.61) | -5.39 - -2.56 | <.001 |
| anxiety symptoms | *M* = 15.02, *SD* = 12.72 | *M* = 11.53, *SD* = 10.96 | -4.58(967.76) | -4.98 - -1.99 | <.001 |

*Note.* CA = childhood adversity. SES = socio-economic status. [*1]We applied Welsh's two-tailed independent sample t-test to account for potentially unequal variances across groups. [*2]As SES was split in five ordered categories, we applied the two-tailed Asymptotic Cochran-Armitage test (Hothorn et al., 2008). [*3]We applied two-tailed Pearson's chi-square tests. [*4]The confidence interval (CI) for the difference in location estimates, corresponding to the alternative hypothesis. [*5]SES was assessed with the ACORN classification system (http://www.caci.co.uk; Morgan & Chinn, 1983). [*6]Psychiatric history was assessed with the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Present and Lifetime Version), at age 14 additionally including learning disabilities, clinical sub-threshold diagnoses and deliberate self-harm, and at age 17 additionally including clinical sub-threshold diagnoses and deliberate self-harm (Kaufman et al., 1997).

### 4.3.2 RF Mean Level Changes

**Group comparisons.** At both age 14 and 17, distress was significantly higher and nine of the ten RFs were significantly lower in the CA+ group (please note, RFs are scored in such a way that higher levels are more protective; see Table 4.3). The tenth RF, reflective rumination, was also significantly lower in the CA+ group, but only at age 17, not at 14. The general pattern clearly indicates that RFs are lower and distress is higher in the CA+ than in the CA- group, during both early and later adolescence.

Table 4.3
*RF and general distress comparisons: CA+ (n = 631) versus CA- (n = 499) groups*

| | Age | CA+ | CA- | *W* / $\chi^2$*(df)* | 95% *CI*[*1] | *p*[*2] |
|---|---|---|---|---|---|---|
| Friendship support (high) | 14 | 0.09 | 0.23 | 173600 | .04 - .22 | <.01 |
| | 17 | 0.07 | 0.30 | 180700 | .12 - .33 | <.001 |
| Family support (high) | 14 | -0.02 | 0.17 | 178690 | .09 - .29 | <.001 |
| | 17 | -0.07 | 0.14 | 180780 | .12 - .33 | <.001 |
| Family cohesion (high) | 14 | -0.10 | 0.29 | 198690 | .30 - .51 | <.001 |
| | 17 | -0.18 | 0.29 | 198080 | .37 - .63 | <.001 |
| Negative self-esteem (low) | 14 | 0.06 | 0.29 | 182270 | .11 - .31 | <.001 |
| | 17 | 0.10 | 0.55 | 187900 | .25 - .58 | <.001 |
| Positive self-esteem (high) | 14 | -0.08 | 0.21 | 188440 | .20 - .41 | <.001 |
| | 17 | -0.14 | 0.22 | 192880 | .26 - .50 | <.001 |
| Ruminative brooding (low) | 14 | 0.03 | 0.19 | 175000 | .07 - .28 | <.01 |
| | 17 | -0.07 | 0.12 | 182540 | .11 - .28 | <.001 |
| Reflective rumination (low) | 14 | 0.10 | 0.20 | 167440 | -.00 - .19 | .066 |
| | 17 | -0.08 | 0.00 | 170430 | .01 - .15 | <.05 |
| Distress tolerance (high) | 14 | -0.06 | 0.25 | 188300 | .21 - .43 | <.001 |
| | 17 | 0.02 | 0.42 | 195600 | .30 - .53 | <.001 |
| Aggression (low) | 14 | low: 498 (s=1)<br>high: 133 (s=0) | low: 440 (s=1)<br>high: 59 (s=0) | 16.27 (1) | | <.001 |
| | 17 | low: 491 (s=1)<br>high: 140 (s=0) | low: 425 (s=1)<br>high: 74 (s=0) | 09.35 (1) | | <.01 |
| Expressive suppression (low) | 14 | low: 418 (s=1)<br>high: 213 (s=0) | low: 371 (s=1)<br>high: 128 (s=0) | 08.31 (1) | | <.01 |
| | 17 | low: 396 (s=1)<br>high: 235 (s=0) | low: 355 (s=1)<br>high: 144 (s=0) | 08.42 (1) | | <.01 |
| General Distress | 14 | -0.09 | -0.40 | 130950 | -.43 - -.18 | <.001 |
| | 17 | -0.09 | -0.68 | 125400 | -.75 - -.38 | <.001 |

*Note.* CA = childhood adversity. All RFs are scored in such a way that high values are protective (e.g. high levels of high friendship support or high levels of low negative self-esteem) and low values are harmful (e.g. low levels of high friendship support or low levels of low negative self-esteem). The continuous general distress variable is scored in such a way that the higher the value the higher the level of general distress. [*1]The confidence interval (CI) for the difference in location estimates, corresponding to the alternative hypothesis. [*2]Please note the p-values are corrected for the false discovery rate, which is why the CIs do not have to contain 0 for the p-value to be nonsignificant.

**Temporal comparisons.** In both groups, two RFs had lower mean levels at age 17 than at age 14: ruminative brooding and reflection. In the CA- group, distress tolerance and negative self-esteem had higher mean levels at age 17 than at age 14. In the CA+ group, only distress tolerance had higher mean levels at age 17 than at age 14. All other RFs did not change significantly over time (see Figure 4.1). Importantly, age-CA interaction effects did not predict the RFs and general distress (see Table 4.4). Therefore, all RFs that changed between age 14 and 17 changed similarly in the two groups.

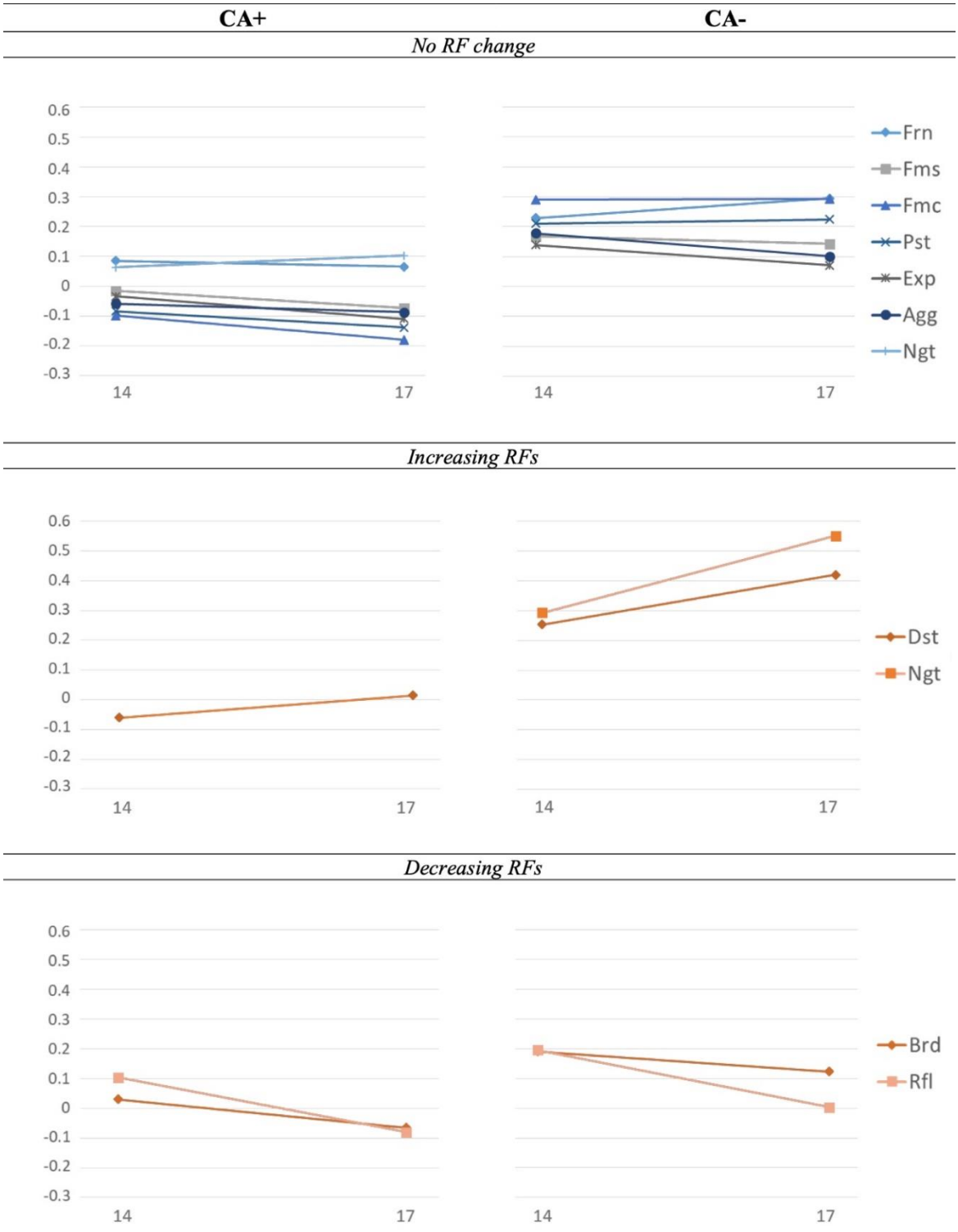


*Figure 4.1.* RF mean level comparisons. CA = childhood adversity. All scores are derived from strongly invariant confirmatory factor analyses. All RFs are scored in such a way that high values are protective (e.g. high levels of high friendship support or high levels of low negative self-esteem) and low values are harmful (e.g. low levels of high friendship support or low levels of low negative self-esteem). **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression.

Table 4.4
*RF and general distress comparisons: age 14 versus age 17*

| | CA | Age 14 | Age 17 | *V* | *95% CI*[*1] | *p*[*2] | age*x*CA[*3] | age*x*CA *p* |
|---|---|---|---|---|---|---|---|---|
| Friendship support (high) | yes | 0.09 | 0.07 | 102800 | -.04 - .08 | .55 | -.09 | .63 |
| | no | 0.23 | 0.30 | 55837 | -.13 - -.00 | .08 | | |
| Family support (high) | yes | -0.02 | -0.07 | 109330 | .00 - .12 | .07 | -.03 | .81 |
| | no | 0.17 | 0.14 | 64965 | -.03 - .09 | .49 | | |
| Family cohesion (high) | yes | -0.10 | -0.18 | 110280 | .01 - .14 | .06 | -.08 | .63 |
| | no | 0.29 | 0.29 | 61400 | -.08 - .06 | .76 | | |
| Negative self-esteem (low) | yes | 0.06 | 0.10 | 90292 | -.19 - -.01 | .07 | -.22 | .13 |
| | no | 0.29 | 0.55 | 41185 | -.43 - -.24 | <.001 | | |
| Positive self-esteem (high) | yes | -0.08 | -0.14 | 108460 | -.00 - .11 | .09 | -.07 | .63 |
| | no | 0.21 | 0.23 | 59923 | -.09 - .04 | .49 | | |
| Ruminative brooding (low) | yes | 0.03 | -0.07 | 116300 | .05 - .16 | <.01 | -.03 | .81 |
| | no | 0.19 | 0.12 | 71074 | .02 - .14 | <.05 | | |
| Reflective rumination (low) | yes | 0.10 | -0.08 | 130350 | .14 - .26 | <.001 | .01 | .96 |
| | no | 0.20 | 0.00 | 82603 | .14 - .27 | <.001 | | |
| Distress tolerance (high) | yes | -0.06 | 0.02 | 81643 | -.11 - -.04 | <.001 | -.09 | .63 |
| | no | 0.25 | 0.42 | 36790 | -.20 - -.13 | <.001 | | |
| Aggression (low) | yes | low: 498 (=1)<br>high: 133 (=0) | low: 491 (=1)<br>high: 140 (=0) | 7138 | | .59 | 1.22 | .63 |
| | no | low: 440 (=1)<br>high: 59 (=0) | low: 425 (=1)<br>high: 74 (=0) | 2438 | | .18 | | |
| Expressive suppression (low) | yes | low: 418 (=1)<br>high: 213 (=0) | low: 396 (=1)<br>high: 235 (=0) | 9333 | | .14 | 1.01 | .96 |
| | no | low: 371 (=1)<br>high: 128 (=0) | low: 355 (=1)<br>high: 144 (=0) | 4375 | | .21 | | |
| General Distress | yes | -0.09 | -0.09 | 106940 | -.02 - .22 | .14 | .27 | .13 |
| | no | -0.40 | -0.68 | 79608 | .22 - .46 | <.001 | | |

*Note.* CA = childhood adversity. All RFs are scored in such a way that high values are protective (e.g. high levels of high friendship support or high levels of low negative self-esteem) and low values are harmful (e.g. low levels of high friendship support or low levels of low negative self-esteem). The continuous general distress variable is scored in such a way that the higher the value the higher the level of general distress. [*1]The confidence interval (CI) for the difference in location estimates, corresponding to the alternative hypothesis. [*2]Please note the p-values are corrected for the false discovery rate, which is why the CIs do not have to contain 0 for the p-value to be nonsignificant. [*3]For linear models the interaction is reported as *b*-value and for binomial logit models as odds ratio.

### 4.3.3 RF Interrelation Changes

**Group comparisons.** Figure 4.2 depicts the RF networks that are corrected for general distress for the CA+ and the CA- group, as well as for age 14 and 17. For age 14, the CA+ and CA- networks were invariant (M = .14, p = .43). However, the global network expected influence, which indicates the degree to which RFs are positively interrelated, was significantly lower in the CA+ network ($EI_{CA+}$ = 2.27, $EI_{CA-}$ = 2.71, EI = 0.44, p = .02). This suggests that in the CA+ network RFs are less likely to enhance each other than in the CA- network. Four individual RF interrelations differed between the CA+ and the CA- networks (see Appendix C.13). For age 17, both the global network structure invariance and the expected influence comparison tests were not significant (M = .11, p = .86; $EI_{CA+}$ = 2.45, $EI_{CA-}$ = 2.49, EI = 0.04, p = .83). Moreover, only one individual RF interrelation differed between the CA+ and the CA- networks (see Appendix C.13).

**Temporal comparisons.** When we compared the networks between age 14 and 17, the networks were invariant and did not differ in global network expected influence, in both the CA+ (M = .10, p = .73; $EI_{14}$ = 2.27, $EI_{17}$ = 2.45, EI = 0.18, p = .36) and the CA- group (M = .12, p = .76; $EI_{14}$ = 2.71,

$EI_{17}$ = 2.49, EI = 0.22, p = .26). In the CA+ network two individual RF interrelations changed significantly between age 14 and 17, whereas none changed in the CA- network, see Appendix C.13.

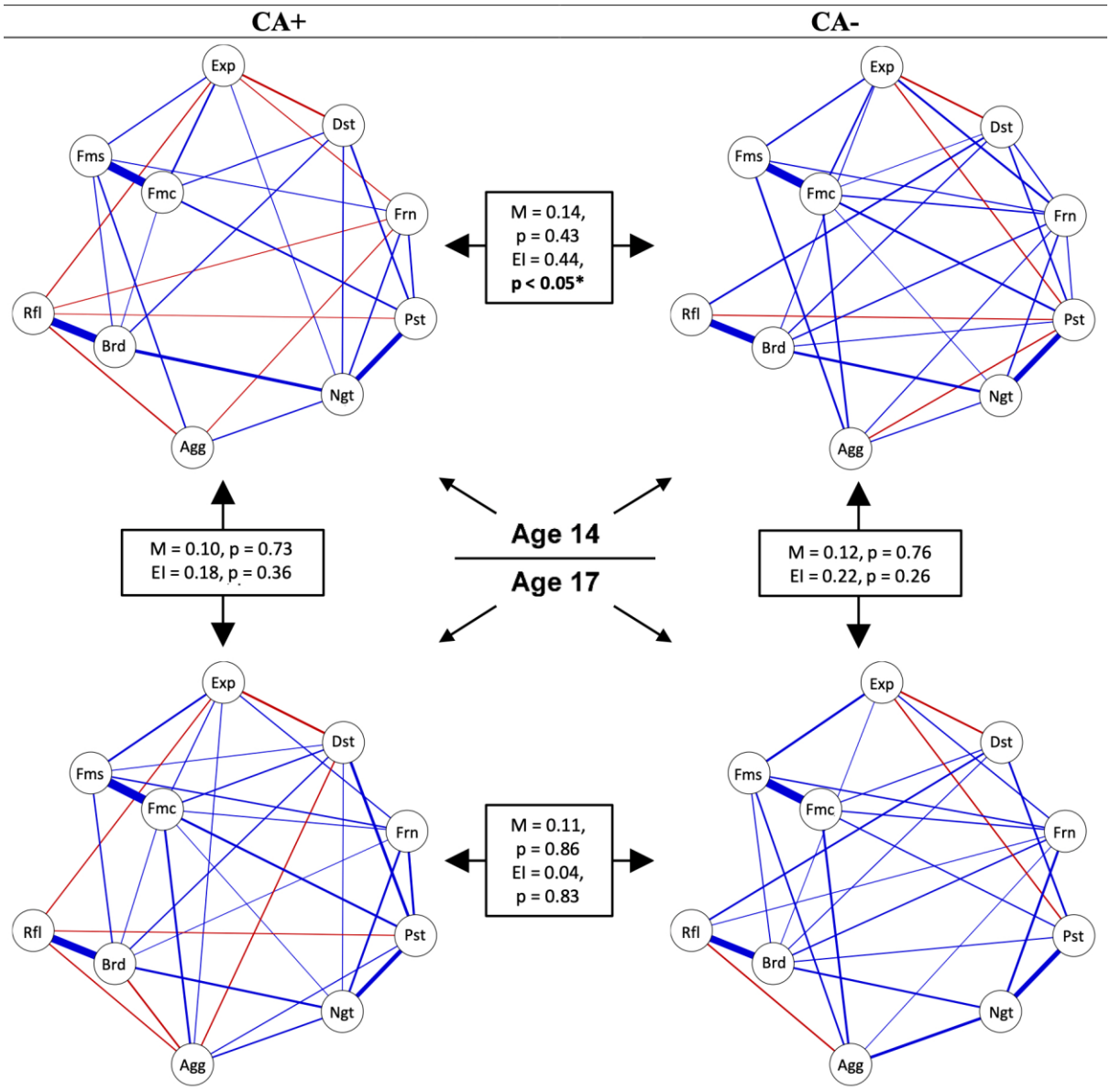



*Figure 4.2.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for age 14 (upper panel) and age 17 (lower panel) corrected for the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples). The above networks with faded interrelations can be found in Appendix C.14. Please note, the upper panel of the Figure is similar to a Figure in a previous report on this sample (see Chapter 3 or Fritz, Fried et al. (2018) in Scientific Reports; can be retrieved from https://doi.org/10.1038/s41598-018-34130-2; information regarding the publishing license of the original Figure, and information regarding differences with the above Figure can be found in Appendix C.15).

### 4.3.4 Changes in Pathways between RFs and General Distress

**Group comparisons.** First, we explored the *direct pathways* between the RFs and general distress (Figure 4.3 upper panel). At age 14, most RFs had negative direct pathways, in both the CA+ and the CA- group, indicating that high RFs go together with low distress (or vice versa). Yet, those *negative direct pathways* to distress did overall not differ in strength between the CA+ and the CA- group ($DP_{CA+}$ = - 1.40, $DP_{CA-}$ = -1.28, DP = 0.12, p = .25; i.e. a more negative DP value indicates a stronger (negative) direct pathway and a less negative DP value indicates a weaker (negative) direct pathway). At age 17, the results were similar as the strength of the *direct pathways* did not differ between the two groups ($DP_{CA+}$ = -1.47, $DP_{CA-}$ = -1.33, DP = 0.15, p = .21). Importantly, the *direct pathway* results do not consider that some RFs have stronger *indirect* than *direct* effects on distress, i.e. via other RFs. To this end, we next calculated *shortest pathways* between RFs and distress, which indicate the quickest way to traverse the network from the RF to distress (Figure 4.3 lower panel). At age 14, the majority of RFs in the CA+ group had a *direct shortest pathway* with general distress (i.e. 6 out of 10), whereas the majority of RFs in the CA- group had an *indirect shortest pathway* with distress (i.e. 6 out of 10). However, the overall strength of the *shortest pathways* did not differ between the two groups ($SP_{CA+}$ = 78.62, $SP_{CA-}$ = 93.42, SP = 14.81, p = .18; i.e. a lower SP value indicates a stronger (and thus shorter) shortest pathway and a higher SP value indicates a weaker (and thus longer) shortest pathway). At age 17, the two groups no longer differed in the number of *negative shortest pathways* and neither in the strength of the *shortest pathways* ($SP_{CA+}$ = 92.13, $SP_{CA-}$ = 93.51, SP = 1.38, p = .93).

**Temporal comparisons.** When comparing the *direct pathways* between the RFs and general distress between age 14 and age 17, no significant temporal differences were found in the CA+ (CA+: $DP_{14}$ = -1.40, $DP_{17}$ = -1.47, DP = 0.07, p = 0.50) and the CA- group ($DP_{14}$ = -1.28, $DP_{17}$ = -1.33, DP = 0.05, p = 0.70). Similarly, when comparing the *shortest pathways* between age 14 and age 17, we again did not find significant temporal differences in the CA+ ($SP_{14}$ = 78.62, $SP_{17}$ = 92.13, SP = 13.52, p = 0.18) and the CA- group ($SP_{14}$ = 93.42, $SP_{17}$ = 93.51, SP = 0.09, p = 0.99).

## 4.4 DISCUSSION

We aimed to shed light on RF changes between age 14 and age 17, and investigated (a) RF mean levels, (b) RF interrelations, and (c) pathways from the RFs to general distress, in adolescents with and without CA. Regarding RF mean levels (a) we found that although inter-personal RFs (e.g. friendships) seemed to stay stable, some intra-personal RFs (e.g. distress tolerance) changed between age 14 and 17. Interestingly, all RFs that in- or decreased between age 14 and 17 changed similarly in the two groups. Moreover, the CA+ group had lower RFs and higher distress at both ages. Regarding RF interrelations (b) we found that at age 14, but not at age 17, RFs were less positively interrelated in the CA+ group. This suggests that the RFs are less likely to enhance each other in the CA+ compared to the CA- network. Regarding RF-distress pathways (c) our results indicate that the strength of the pathways did neither differ between the CA+ and the CA- group, nor over time, suggesting that RFs may be similarly protective in both groups and at both ages. Below I will outline how our findings inform

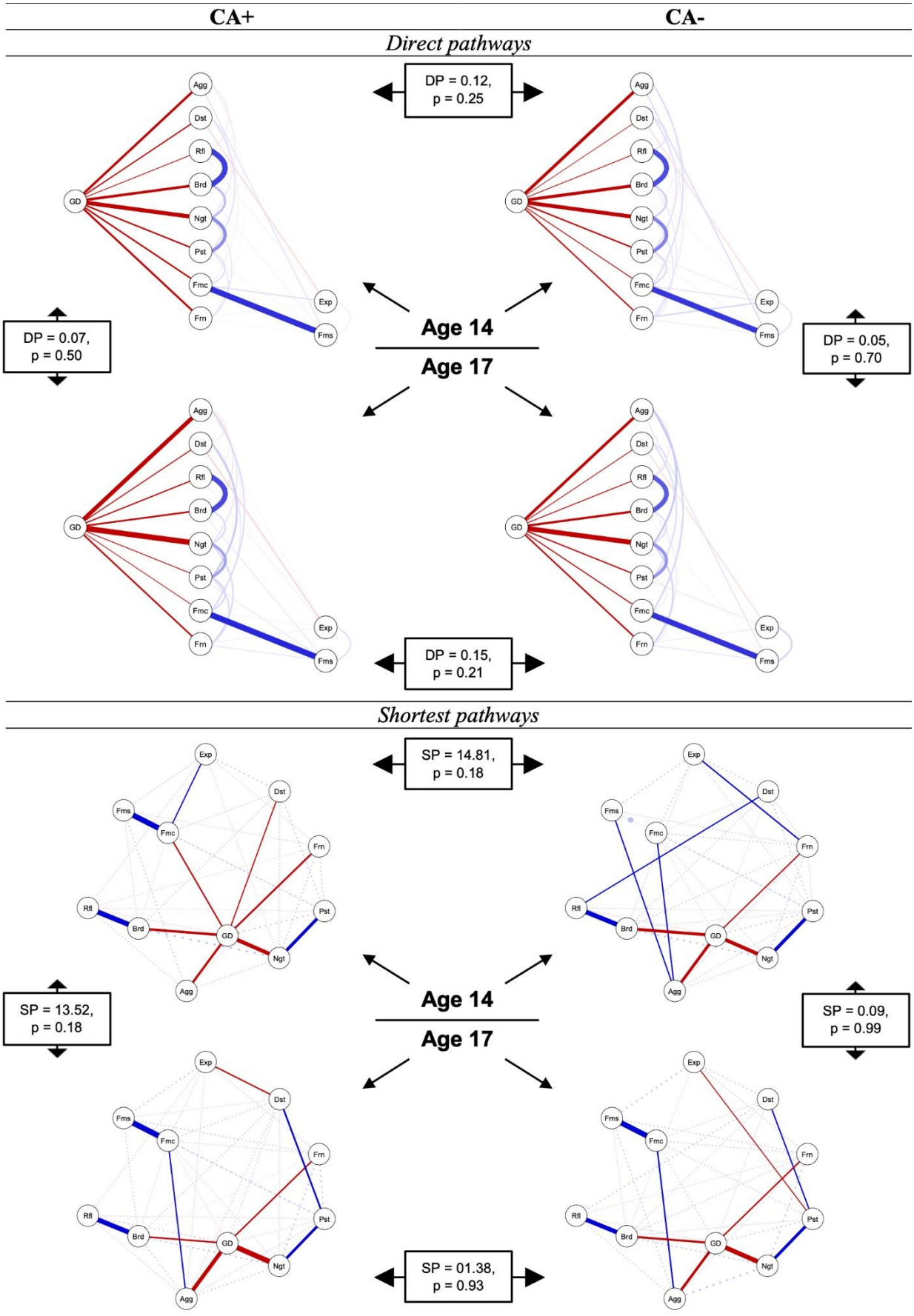



*Figure 4.3.* Direct (DP) and shortest pathways (SP) between the resilience factors (RFs) and the general distress variable, for the CA+ (n = 631) and the CA- (n = 499) group. The upper panel depicts direct and the lower panel

the shortest pathways between the RFs and general distress. Within the panels the upper part depicts the networks for age 14 and the lower part the networks for age 17. Non-transparent lines = direct/shortest pathway of interest. Transparent/dotted lines = all remaining partial regularized correlation relationships. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression. Please note, the upper part of the lower panel is similar to a Figure in a previous report on this sample (see Chapter 3 or Fritz, Fried et al. (2018) Scientific Reports; can be retrieved from https://doi.org/10.1038/s41598-018-34130-2; information regarding the publishing license of the original Figure, and information regarding differences with the above Figure can be found in Appendix C.15).

about the complex nature of RFs and will discuss tentative accounts for why effects associated with CA are not only proximal, but are often found to have a lasting impact on mental health.

### 4.4.1 RF Mean Level Changes

All inter-personal RFs (i.e. friendship support, family support, and family cohesion) seemed to stay stable between age 14 and 17, showing that, in this cohort, adolescents perceive their social support environment to be similar during early and later adolescence. The mean levels of some intra-personal RFs changed however between age 14 and 17 (i.e. distress tolerance, brooding, and reflection in both groups, as well as negative self-esteem in the CA- group). Adolescents reported a higher level of distress tolerance at age 17 than at age 14, which potentially may be explained by the improvement of executive functions and emotion regulation strategies. Previous literature has shown that executive functions, such as inhibitory control which facilitates the regulation of cognition and behaviour, develop and improve until adulthood (Diamond, 2013; Friedman et al., 2016). Similarly, the use of emotion regulation strategies has been found to be significantly lower in mid-adolescence (age 15) than in young adulthood (age 19; Zimmermann & Iwanski, 2014).

In the literature, findings regarding changes in rumination are mixed. For example, Zimmerman and Iwanski (2014) did not find a significant difference in rumination between age 13 and 17, whereas Frydenberg and Lewis (2000) showed that ruminative worrying is higher at age 16 than at age 14. In line with Frydenberg and Lewis (2000), our sample reported higher (more harmful) levels of reflective rumination and ruminative brooding at age 17 than at age 14. Besides the increase in rumination, our CA- group reported a decrease in negative self-esteem between age 14 and 17. Those results together suggest that although CA- adolescents may worry and reflect more about their experiences and behaviours during later adolescence, they may not attach those negative thoughts and evaluations to their self-image. Despite the fact that there was no significant decrease in negative self-esteem in the CA+ group, the change in negative self-esteem from age 14 to 17 did not differ significantly between the two groups. While further replication of our results is required, our results suggest that between early and later adolescence mechanisms emerge that alter the perception of the self (e.g. negative self-esteem, rumination) and self-regulation (e.g. distress tolerance, rumination; Diamond, 2013; Friedman et al., 2016; Frydenberg & Lewis, 2000; Kim et al., 2015; Zimmermann & Iwanski, 2014).

Our results further showed that all changes in RF mean levels between early and later adolescence were similar in the CA+ and the CA- groups. Crucially, however, the CA+ group had lower RFs at both ages, which is in line with previous research (Almquist et al., 2018). Hence, CA does not

seem to inhibit RF changes, but seems to increase the risk of persistently lower RFs. Those findings support the hypothesis that lower and therefore possibly disadvantageous RF levels after CA are transferred forward from early to later adolescence (Kim et al., 2015; McLaughlin, 2016), which underpins the importance of revealing which factors and processes lend themselves best to aid optimal development after CA (Kim et al., 2015; McLaughlin, 2016).

In sum, our findings show that individual RFs change differently between early and later adolescence, but that the change pattern is similar in groups of CA+ and CA- adolescents. Based on those results I cautiously suggest implications for future research, while reminding the reader that our findings only allow for group level not individual level conclusions. The main questions that arise from our mean level findings are threefold. Firstly, one could ask whether RFs that seem to increase naturally during adolescence (e.g. distress tolerance) are particularly amenable and therefore more efficient intervention targets for reducing distress. Similarly, one may wonder whether it may be as advantageous to intervene on worsening RFs (e.g. rumination), to reduce or prevent such a decline. Regarding RFs that stay stable (e.g. friendships, family support and family cohesion), the arising question seems different. Stable RF levels may be advantageous for adolescents with a high level of those RFs, but may be disadvantageous for adolescents with a persistently low level of those RFs. Speculatively, stable RFs may function as a "vulnerability marker" when being persistently low, and early detection may be beneficial. Replication studies and translational research are crucially needed to answer these important questions, as such knowledge may eventually shed light on which RFs should be targeted in order to aid successful mental health development in adolescents with and without CA.

### 4.4.2 RF Interrelation Changes

Despite the fact that the RF levels differed between the CA+ and the CA- group at both age 14 and 17, RF interrelations differed between the two groups only at age 14, not at age 17. This suggests that CA may have a more pronounced effect at age 14, as it then goes together with both differential RF levels and differential RF interrelations. One account could be proximity of CA, as CA was measured up to the age of 14. This would be in line with previous work suggesting that although CA has deleterious effects on mental health across the life course, it has a particularly strong effect on a shorter term and accordingly a decreasing effect on affective and behaviour disorders from childhood to young adulthood (Kessler et al., 2010; Shanahan, Copeland, Costello, & Angold, 2011).

Interestingly, on a *global* network structure level, taking the overall pattern of RF interrelations into account, both the CA+ and the CA- network were invariant between early and later adolescence. Moreover, neither the CA+ nor the CA- network changed in the degree to which RFs are expected to enhance each other (i.e. expected influence) between early and later adolescence. I believe that the lack of temporal changes on the *global* network level is unlikely to be explained by power, as we did detect a difference in expected influence in other comparisons (see example in the next paragraph). Moreover, on the *local* network structure level, we also identified only minor changes between early and later adolescence. In the CA+ network one out of 45 possible RF interrelations turned more positive and one turned less positive between age 14 and 17 (see Appendix C.13), which may have cancelled

each other out and thus may help explain why there was little change in the expected influence of the CA+ network. In the CA- network none of the 45 RF interrelations changed significantly between age 14 and 17 (see Appendix C.13). Hence, those findings point towards a general stability of RF interrelations between early and later adolescence, in both the CA+ and the CA- network. If this would generalize to other cohorts, it may offer one account for the finding that CA often has lasting effects on mental health (Greif Green et al., 2010; Raposo, Mackenzie, Henriksen, & Afifi, 2014).

Of note, those findings were slightly different for the RF networks which are not corrected for general distress (see Appendix C.7), as those networks differed in positive connectivity between age 14 and age 17 in the CA+ group. At age 17 the CA+ network was significantly more positively interrelated than at age 14. This finding suggests that in the CA+ (not the CA-) group there is some improvement in the degree to which RFs can potentially enhance each other, between early and later adolescence. Yet, as this finding does not hold when we take general distress into account, the effect should be considered with caution.

For both the CA+ and the CA- network, at both age 14 and age 17, the family, brooding and negative self-esteem RFs were most positively connected with the other RFs (for more details see Appendix C.10). Hence, those RFs are potentially important in driving the positive connectivity of the RF networks and in underpinning the degree to which RFs can enhance each other. Interestingly, in terms of mean levels the family RFs stayed stable in both groups, the brooding RF decreased in both groups and the negative self-esteem RF increased in the CA- group between age 14 and age 17. This suggests that (changes in) mean levels of RFs may not, or at least not directly, impact the degree to which the RFs can enhance other RFs. Thus, our RF mean level and RF network model analyses provide independent but complementary insights. To further improve knowledge about the clinical relevance of those indicators, future research needs to examine whether RF mean levels or RF interrelations characteristics (such as *expected influence* coefficients) are better predictors for subsequent mental health. Such knowledge needs to be obtained before our network findings can inform clinical research, as knowledge on the prediction magnitude is essential for picking promising RF targets for translational studies.

### 4.4.3 Changes in Pathways between RFs and General Distress

Our findings showed that most RFs had direct negative pathways with distress, in both the CA+ and the CA- group, indicating that high RFs decrease distress, high distress decreases RFs, or both mutually influence each other. As all investigated RFs have empirically been shown to significantly decrease subsequent distress (see Chapter 2), it seems plausible that RF-distress pathways may not only over time, but also concurrently, operate as protective pathways. In the same vein it is however also plausible that high distress reduces the protective effects of RFs (concurrently and/or over time). Such mutualistic coupling effects (e.g. Kievit et al., 2017) need to be examined in future research. At both age 14 and 17 those potentially protective pathways appeared to be similarly strong in the two groups, regardless of solely investigating *direct* or also *indirect* pathways (i.e. via other RFs). Moreover, we did not detect differences between age 14 and 17, suggesting that RF-distress pathways seem stable between age 14 and 17.

Importantly however, when taking our mean level findings into account ─ i.e. that the CA+ group had lower RFs and higher distress than the CA- group ─ a more elaborate interpretation emerges. That is, despite the fact that RF-distress pathways seem on the first glance to be similarly protective in the two groups, the combination of lower RFs and higher distress in the CA+ group supports the notion that RF-distress pathways operate on a different, and presumably more disadvantageous, mean level than in the CA- group. As lower RFs, higher distress, and potentially disadvantageous RF-distress pathways seemed to be rather stable from early to later adolescence, this may be another account for why exposure to CA is frequently found to not only have a short-term, but also a longer lasting impact on mental health (Greif Green et al., 2010; Raposo et al., 2014).

The four RFs that were most strongly interrelated with distress, in both the direct and the shortest pathway models, were negative self-esteem, brooding, aggression and friendship support. Interestingly, the first two of those RFs were also among the RFs being most positively connected with the other RFs, in both groups and at both ages. Hence, if replication of our findings would hold, the negative self-esteem and brooding RFs may be of particular interest for future prediction studies, as they not only seem to have the highest potential of increasing other RFs, but also seem to have the highest potential in reducing distress, and therefore may also have a high potential in reducing subsequent mental health problems.

### 4.4.4 Limitations

Our research has several limitations. First, CA was assessed with retrospective caregiver report, which may be inaccurate due to for example limited recall, limited knowledge, or embarrassment. To enhance recall caregivers were encouraged to use assisting material (e.g. photo albums; Dunn et al., 2011), and an event timeline (with the following time windows: 0-5, 5-11, 11-14) was established. Second, the family support and family cohesion RFs were derived from one questionnaire, which may have resulted in more similar response patterns in those RFs. The same argument goes for rumination (reflection and brooding) and self-esteem (high positive and low negative self-esteem) RFs. Third, to enable RF comparisons over time, we had to equate multiple LCFA parameters between age 14 and age 17. This may disadvantage the model accuracy and therefore potentially increase bias in the resulting factor scores. To circumvent this limitation as best we could, we used the least restricted models possible to still meet the assumptions of the respective network and mean change analyses. However, this meant that we could not use the exact same factor scores for the network and the mean change analyses. For completeness, we re-ran the mean change analyses with factor scores derived from the LCFAs that we used for the network analyses (see Appendix C.6). Fourth, we interpret negative interrelations between RFs in networks that take general distress into account as disadvantageous. However, as our models are undirected, we cannot disentangle whether the general distress variable behaved as intended as a confounder, or against our expectation as a collider (Elwert & Winship, 2014), falsely inducing or enhancing these interrelations (for a detailed discussion see Appendix B.14). Fifth, we performed the network models with regularized partial correlations, which currently is the default method. However, recently, other approaches have been suggested such as non-regularized methods (Williams, Rhemtulla, Wysocki, & Rast, 2019). Future research will need to show which methods tend to be most

suitable for psychometric network models. Sixth, as our study contains two time points, we cannot draw conclusions with regard to tipping points or specifically sensitive periods. Likewise, we cannot examine how RFs change from prior to post CA, as we did not assess the RFs prior to CA. Seventh, we used imputation methods to include participants with missing information. Yet, when we pooled the factor model results for the imputed data sets together, we revealed for some models a negative pooled chi-square. As relative fit indices cannot be calculated based on a negative chi-square, the chi-squares had to be set to zero, resulting in arbitrary chi-square dependent ("relative") pooled fit indices. To enable the reader to judge the various models (i.e. being based on the different imputed data sets), we provide a chi-square independent ("absolute") fit index pooled over the separate models (i.e. the standardized root mean residual) and provide chi-square dependent ("relative") fit indices separately for the models. Eighth, it would have been valuable to explore gender effects (Stochl et al., 2019), however, for many of the analyses we may not have had enough power to split the sample additionally with regard to gender. Ninth, the ROOTS participants had on average a slightly higher SES than the average UK population and generalizations may therefore be most valid for above average SES populations (Goodyer et al., 2010).

Regarding the question whether resilience and risk factors are opposing sides of the same coin, the quick, but insufficient answer for our study is probably that many (or most) of the investigated RFs are indeed the flip side of risk factors. For example, self-esteem (or a positive self-concept) is commonly defined as RF and has been discussed as such by many of the seminal resilience researchers, including Michael Rutter, Emmy Werner, Ann Masten, and Michael Ungar (for a review see e.g. Shean, 2015). Yet, at the same time a low level of self-esteem or self-worth is part of the DSM 5 criteria for depression ("Feelings of worthlessness"; American Psychiatric Association, 2013). Hence, whereas a high level of self-esteem may protect against low mood levels, low self-esteem is assumed to contribute to or reflect low mood. As doing this question fully justice is out of the scope of this discussion, I added a more detailed debate on the question to Appendix C.16. Importantly however, regardless of whether resilience and risk factors operate on the same continuum or are inversely correlated but not identical, understanding the nature of RFs seems to have universal appeal as it focuses on what promotes good mental health rather than on what increases mental health problems.

### 4.4.5 Conclusion

Our results support several prior conjectures regarding changes in RF mean levels, for example that lower and therefore disadvantageous levels of RFs are likely to be carried forward over time in adolescents with prior exposure to CA. Our findings also contribute novel hypotheses: for example, they suggest that RF changes are similar in adolescents with and without CA, and that inter-personal mean levels may stay stable, whereas some intra-personal RFs change between early and later adolescence. On a network level CA seemed to have a stronger proximal effect, as RF interrelations differed between the two groups at age 14, but not at age 17.

RF-distress pathways seemed to have similarly protective strengths in both groups, during early and later adolescence. Yet, as RFs are lower and distress is higher in the CA+ group, I cautiously suggest that RF-distress pathways may overall be less advantageous than in the CA- group. As lower

RFs, higher distress, and potentially disadvantaged pathways between RFs and distress seemed to be carried forward from early to later adolescence, our findings may help explain why exposure to CA is frequently found to have a lasting impact on mental health. To pinpoint the clinical relevance of our findings, I recommend future research to examine whether (a) RF mean levels, (b) RF interrelations coefficients, or (c) RFs that score high on both indicators offer the best prediction for subsequent mental health and thus lend themselves best for formulating translational hypotheses. In sum, our study not only sheds light on the complex nature and changes of ten empirically supported RFs between early and later adolescence, but also offers tentative accounts for why CA has strong proximal effects, and is often found to have a lasting impact on mental health.

In the next chapter, I aim to shed some light on the predictive value with which RFs at age 14 reduce mental health problems at age 17. More specifically, I shall try to disentangle to what degree RFs can explain subsequent mental health problems, which RFs are the best indicators for subsequent mental health problems, and with what accuracy RFs can predict subsequent mental health problems.

# CHAPTER 5

# Embracing the Positive: An Examination of How Well Resilience Factors at Age 14 Can Predict Distress at Age 17

Every year, about 1 in 5 people experience mental disorders (Tice et al., 2018; World Health Organization, 2015), of which the most prevalent mental illnesses are depressive and anxiety disorders (World Health Organization, 2015). Half of such mental illnesses first emerge during adolescence (World Health Organization, 2018). About 1 in 3 adolescents have an episode of an anxiety disorder and more than 1 in 10 an episode of a mood disorder, between the ages of 13 and 18 (Ries Merikangas et al., 2010). The prevalence of anxiety disorders tends to remain stable during adolescence, however, mood disorders double between the ages of 13 and 18 (Ries Merikangas et al., 2010). Hence, adolescence seems to be a particularly sensitive time period for the emergence of mental health problems and it is therefore imperative to characterize and predict such vulnerability to psychopathological distress properly.

A growing number of studies has developed screening tools and risk prediction models – also known as risk calculators – for mental health problems (Lawrie, Fletcher-Watson, Whalley, & McIntosh, 2019; Shatte, Hutchinson, & Teague, 2019). For example, Dinga and colleagues (2018) have shown that, among a large variety of psychological and biological variables, only mood severity predicted subsequent depressive symptomology significantly. Still, their prediction model revealed an acceptable accuracy (Dinga et al., 2018). Similarly, Lewis and colleagues (2019) have shown that a constellation of demographics, psychopathology symptoms (i.e. psychotic and internalizing symptoms), and adversity variables can together satisfactorily predict whether adolescents develop post-traumatic stress disorder, following trauma exposure. In a recent systematic review, summarizing literature on mental health screening tools and risk models, 60 studies were identified for depression related diagnoses, 13 for psychopathological stress, five for anxiety related diagnoses, and five for well-being (Shatte et al., 2019). Importantly, the majority of those studies used symptom-related (e.g. questionnaires and interviews), demographical (e.g. adverse life-events), or biological indicators (e.g. inflammatory markers, cortisol, metabolic syndrome, brain-derived neurotrophic factor, white and grey matter, and heart rate variables; Dinga et al., 2018; Lawrie et al., 2019; Pintelas, Kotsilieris, Livieris, & Pintelas, 2018; Shatte et al., 2019). Thus, previous studies primarily examined predictors that are relatively static (e.g. ethnicity or gray matter) and/or risk factors that increase the development of mental health problems (e.g. negative life-events or prior psychiatric symptoms).

Focussing on static and risk factors, however, is only half the story, as it fails to address factors that are amenable and promote mental health. The resilience literature has already identified various factors that are associated with improved subsequent mental health (Afifi & MacMillan, 2011; Braithwaite et al., 2017; Marriott et al., 2014; Traub & Boynton-Jarrett, 2017; Wright et al., 2013), which seem to be overlooked in the development of screening tools and risk calculators. A notable exception is the study of Chen, Huang, and Zhang (2015) in which self-esteem was used to predict subsequent anxiety. Another important exception is the recent study of Meehan and colleagues (2020), which

included alongside various risk indicators four potential resilience factors (sibling warmth, adult involvement, social cohesion and status among peers), to predict internalizing and externalizing disorders following victimization.

Here, my colleagues and I aim to extend the existing prediction literature in several ways. Firstly, we use resilience factors (RFs) as predictors, i.e. factors that have been found to reduce the risk of psychological distress following adverse experiences (see Chapter 2 or Fritz, de Graaff, et al., 2018). We derived the RFs that we study here from the preregistered systematic review in Chapter 2, in which RFs were defined as those factors that moderate and/or mediate the relationship between childhood adversity and subsequent mental health problems. In the resilience literature there is a sparse but ongoing discourse about whether resilience and risk factors are opposing sides of the same coin (for a detailed discussion see Appendix C.16). Some RFs and risk factors seem indeed to be on opposing sides of the same continuum (e.g. RF = high friendship support & risk factor = low friendship support; van Harmelen et al., 2016), whereas for others this apparent dichotomy seems more complex. For example, *high* rumination can be both an RF and a risk factor depending whether its content is positive or negative (e.g. RF = high positive rumination & risk factor = low positive ruminations; RF = low negative rumination & risk factor = high negative rumination; while high positive and high negative rumination often go together; Harding & Mezulis, 2017). Importantly, regardless of whether resilience and risk factors operate on the same continuum, studying the predictive value of RFs has universal appeal as it focuses on what promotes good mental health rather than on what increases mental health problems (see Appendix C.16).

Secondly, we extend the existing literature through focusing exclusively on factors that are amenable to psychotherapeutic change, which is in contrast to the majority of the above reviewed studies, as those mainly focused on relatively static demographic (e.g. ethnicity) and biological (e.g. grey or white matter volume) predictors. More specifically, we predict psychopathological distress from 10 amenable RFs. Three of those RFs operate on an *inter*-individual level: friendship support, family support and family cohesion; and seven on an *intra*-individual level: high positive self-esteem, low negative self-esteem, low brooding, low ruminative reflection, high distress tolerance, a low aggression potential and low expressive suppression (see Chapter 2). Importantly, all those RFs on their own have been found to decrease subsequent mental health problems, yet, research investigating multiple RFs at the same time is so far scarce (Diehl et al., 2012; Scheffer et al., 2018). Recently, my colleagues and I found that these RFs reduce concurrent psychopathological distress with a similar degree in adolescents with and without prior exposure to adversity (see Chapter 4 or Fritz et al., 2019). Moreover, we have shown that the RFs interrelate strongly and can be described as a complex interacting system (see Chapter 3 or Fritz, Fried, et al., 2018)**.** This supports the notion that models that succeed in taking all those factors into account may ecologically be more valid and may successfully reveal those RFs that are particularly important in reducing the risk of mental health problems.

Recently, research has also shed light on the benefits of describing mental health problems as distress continua rather than as discrete diagnosis specific constructs. For example, several studies show that modelling psychopathological symptoms as a continuous latent factor captures a wide range of mental health symptomatology, in terms of both severity and breadth of symptomatology (Brodbeck

et al., 2011; Caspi et al., 2014; Ronald, 2019; St Clair et al., 2017; Stochl et al., 2015), and even seems to generalize well to other disorders (Ronald, 2019). Therefore, such latent continuous constructs may be particularly informative for transdiagnostic prevention and intervention research. Moreover, hybrid models have been developed that describe mental health symptoms as a continuous latent factor and then add categorical classes to the latent factor that differentiate between subgroups on the latent mental distress continuum (e.g. as defined by differences in the distress severity; Clark et al., 2013). Categorical distress scores derived from those models may be particular useful for prediction purposes, as they allow for the estimation of predictive sensitivity and specificity, while taking into account the continuous nature of distress. Yet, to the best of our knowledge, transdiagnostic distress indices have so far rarely been used for predictive purposes and is therefore the third way in which we extend the existing literature.

In sum, we aim to extend the existing literature (a) by using resilience factors rather than risk markers as predictors for subsequent psychopathology, (b) by using amenable (i.e. social, emotional, cognitive and behavioural) rather than static variables (e.g. ethnicity or biological predispositions) as predictors, and (c) by using transdiagnostic distress indices rather than discrete diagnosis specific variables as outcome variables. To this end, we use data from the ROOTS population cohort (n = 1130; Goodyer et al., 2010) to predict distress at age 17 from RFs assessed at age 14, covering the adolescent period during which about half of all mental illnesses start emerging. Given the powerful predictive effects of past mental distress, we evaluate in addition to the relative effects of RFs also the relative effect of distress at age 14 when predicting distress at age 17. A cascade of studies has shown that childhood adversity (CA) vastly increases the risk for mental health problems during adolescence and adulthood (Greif Green et al., 2010; Kessler et al., 2010; McLaughlin, 2016; McLaughlin et al., 2012). Therefore, throughout all analyses, we take the effect of CA before the age of 14 into account. Additionally, we control for gender effects, as being female has frequently been found to increase the risk for distress (e.g. St Clair et al., 2017). In sum, we aim to examine:

a) to what degree RFs can explain subsequent distress,
b) which RFs are the best indicators for subsequent distress, and
c) with what accuracy RFs can predict distress levels three years later.

## 5.2 METHODS

### 5.2.1 Design

The ROOTS study is a population cohort for which 1238 adolescents were recruited at age 14 and reassessed at age 17. The adolescents were recruited in 2005 and 2006, via 18 schools in and around Cambridgeshire. The adolescents and one parent had to provide written informed consent. ROOTS was approved by the Cambridgeshire Research Ethics Committee (03/302) and was conducted along the lines of Good Clinical Practice guidelines and the Declaration of Helsinki (Goodyer et al., 2010).

### 5.2.2 Sample

Here we included all adolescents who had data for potential CA experiences (CA+: n = 638; CA-: n = 501) and had less than 85% missingness on the analyses variables (n = 1188). Prior to the main analyses we imputed missing data and could therefore eventually analyse data of 1130 adolescents, of which 631 with and 499 without prior exposure to CA, and of which 620 were female and 510 male.

### 5.2.3 Childhood Adversity

CA was assessed with the Cambridge Early Experiences Interview (CAMEEI), which is a semi-structured interview performed with the primary carer (Dunn et al., 2011). CAs were defined as adverse experiences or severely stressful events that happened between birth and the age of 14. The assessed CAs include a wide range of intra-family events/experiences (e.g. sexual, physical or emotional maltreatments, or parental mental illness), but also cover external events (e.g. a fire or exposure to war). For a detailed description see Dunn and colleagues (2011). These authors clustered the adolescents based on their CA experiences into four latent classes (i.e. no, moderate, severe and atypical parenting CA), separately for the time periods early (age 0 to 5), middle (age 5 to 11) and late childhood (age 11 to 14; Dunn et al., 2011). As in Chapter 3 and 4, we dichotomized the CA variable in CA+, which is “moderate, severe and/or atypical parenting CA” for at least one of the three time periods, and CA-, which is “no CA” for any of the three time periods.

### 5.2.4 Distress

At age 14 and 17, distress was assessed with 41 items of which 28 had a focus on anxiety symptoms (Revised Children’s Manifest Anxiety Scale; Reynolds & Richmond, 1978) and 13 a focus on depressive symptoms (Short Mood and Feelings Questionnaire; Messer et al., 1995).

### 5.2.5 Resilience Factors

In accordance with Chapter 3 and 4, we investigated 10 RFs that were identified in our preregistered systematic review in Chapter 2, and were assessed in ROOTS (Goodyer et al., 2010). All RFs were assessed at age 14:

1. Friendship support: five items of the Cambridge Friendships Questionnaire (Goodyer et al., 1989).
2. Family support: five items of the McMaster Family Assessment Device (Epstein et al., 1983).
3. Family cohesion/climate: seven items of the McMaster Family Assessment Device. For brevity I write family cohesion throughout the Chapter (Epstein et al., 1983).
4. Positive self-esteem: five items of the Rosenberg self-esteem scale (Rosenberg, 1965).
5. Negative self-esteem: five remaining items of the Rosenberg self-esteem scale (of note, the items are reversed; Rosenberg, 1965).
6. Reflective rumination: five items of the Ruminative Response Scale (RRS; of note, the items are reversed; Burwell & Shirk, 2007; Treynor et al., 2003).
7. Ruminative brooding: five items of the RRS (of note, the items are reversed; Burwell & Shirk, 2007; Treynor et al., 2003).

8. Aggression: four items of the Behaviour Checklist (11 questions based on the DSM-IV criteria for conduct problems; of note, the items are reversed; American Psychiatric Association, 2000; Goodyer et al., 2011).
9. Distress tolerance: five items of the Emotionality Activity Sociability Temperament Survey (Bould et al., 2013).
10. Expressive suppression*:* one item of the Antisocial Process Screening Device (of note, the item is reversed; Poythress et al., 2006).

Items of five RFs had to be reversed to ensure that all RFs are scored in such a way that high values are protective. The first eight RFs are based on self-report, and the last two on parent report. More detailed information regarding the exact content of the RF constructs can be found in Chapter 3. The psychometrics of the RF measures are described in Appendix B.2.

### 5.2.6 Analyses

**Data imputation.** Prior to the main analyses we imputed missing data. Most participants with missing data had missingness at age 17. Yet, some adolescents had missing data at age 14, and others had just incidentally missing items at age 14 and/or 17. Details can be found in Appendix C.4 Table C.2. Overall, missingness on the RFs and general distress could to some degree be explained by exposure to CA, gender, affective symptoms, and a prior psychiatric history (see Appendix C.4 Table C.3). We used multivariate multiple imputation methods to estimate 10 complete data sets with estimated scores for the missing data. For data with more than two categories we used predictive mean matching algorithms and for binary data logistic regression. To enhance the imputation model accuracy, we included 103 items measuring the RFs, 122 items measuring anxiety and depression symptoms, and seven explanatory variables (CA, gender, socio-economic status, prior psychiatric history, and age at occasion 1 and 2 – for measurement details see Table 5.1), leading to a total of 232 items. We did not impute information for the CA factor as not all CA experiences may be adequately predictable (e.g. a car crash). In sum, we were able to estimate data for 1188 participants.

**Variable estimation.** We computed the RFs based on unidimensional confirmatory factor analyses (CFAs; except for expressive suppression as this was assessed with only one item). We use factor scores and not sum scores to evade tau-equivalence and to decrease measurement error as much as possible (for a rationale and explanation see Appendix C.5 Part A). As all items ranged between three and six answer categories, we used categorical CFAs with a weighted least square mean and variance adjusted (WLSMV) estimator. The distress factor was similarly estimated using a longitudinal, unidimensional, categorical CFA (also with the WLSMV estimator), and was identified according to the strongly invariant model described by Wu and Estabrook (2016; for a more detailed rationale see Appendix D.1). We estimated all CFAs for the 1188 participants and pooled the results across the 10 imputation sets. We then extracted the factor scores, that were pooled over the 10 imputation analyses, and used those for the main analyses. For the main analyses we could include 1130 of the 1188 participants, as those had assessed information for CA. For completeness, we performed all analyses also on non-imputed data which can be found in online Supplement II.

**Analyses.** We set out to conduct three sets of analyses, to answer the three research questions: (1) to what degree can RFs (and/or distress) explain subsequent distress, (2) which RFs are the best indicators for subsequent distress (with and without prior distress being taken into account), and (3) with what accuracy can RFs (and/or distress) predict subsequent distress. The first two sets have a predominant focus on "explanation", to better understand the relationships of RFs (and/or distress) at age 14 with subsequent distress at age 17. The last set has a predominant focus on classical "prediction", to answer the question how well RFs (and/or distress) at age 14 can predict an adolescent's subsequent distress level at age 17. That is, the last set of prediction models had the aim to predict an adolescent's distress level at age 17 solely from the knowledge on RFs (and/or distress) at age 14, and then compare the predicted distress score back to the actually observed distress score. Statistically speaking, the analytic techniques for both the explanation-focussed and the prediction-focussed analyses were derived from a regression-based framework, but their theoretical focus differed. As such, the explanation- and prediction-focussed analyses can provide different, but complementary insights. To put it in Yarkoni and Westfall's words (2017; p. 2) "an emphasis on prediction can be viewed not as an opponent of explanation but rather as a complementary goal that can ultimately increase theoretical understanding"; and I would argue that for my specific research question it has the particular additional advantage of providing novel knowledge for translational research on risk and mental health screening. Below I will explain the analyses, including the respective methodological procedures and modelling strategies, that I conducted to answer the three research questions.

First, we performed a series of multiple linear regressions to find out to what degree RFs (and/or distress) at age 14 can explain distress at age 17. More specifically, those analyses were performed to examine the directionality of the regressors (i.e. +/- sign of the b-values) and to investigate whether RFs and distress at age 14 add (a significant amount of) variance to the explanation of distress at age 17. The first two models functioned as baseline models, one only included CA (model B1) and the other one included CA and gender as regressors (model B2). The next three models were the main models of interest: All contained CA and gender as regressor, the first model additionally contained the ten RFs (model M1), the second model additionally contained distress at age 14 (model M2), and the third model additionally contained both the RFs and distress at age 14 (model M3). We compared the models against each other using Likelihood-Ratio tests. We conducted the three main models mostly for two reasons. Firstly, we tested whether both the RFs (M1) and age-14 distress (M2) explained subsequent age-17 distress significantly. This seemed important, as the explanatory effect of previous distress on future distress has often been investigated, but less is known about the explanatory effect of the RFs (and particularly not of multiple RFs in conjunction). Secondly, we aimed to explore the effect of the RFs on age-17 distress over and above the effect of age-14 distress (M3), as this shows (a) whether RFs explain (a significant amount of) variance in age-17 distress in addition to the variance explained by age 14-distress, and thereby (b) also gives an indication for whether RFs can explain change in distress between age 14 and 17. Moreover, we re-estimated the models separately for the CA+ and the CA- groups as well as for males and females, to explore group effects.

Second, we performed a series of regression-based relative importance analyses to examine which RFs at age 14 are the best indicators for distress at age 17 (with and without age-14 distress

being taken into account). More specifically, we aimed to disentangle the relative importance (RI) of the regressors in explaining general distress at age 17. Disentangling the RIs is of particular importance when the regressors are (or are assumed to be) strongly correlated, as every order of regressors then results in a different decomposition of sum of squares (Groemping, 2006). Here, we examined the RI metric "lmg" (cf. Lindeman, Merenda, & Gold, 1980) which calculates sequential $R^2$s while permuting and then averaging over the regressor orders (Groemping, 2006). To this end, we performed the three above described main models (M1, M2, and M3) as RI analyses. We conducted the three models mostly for two reasons. Firstly, estimating the relative importance of the 10 RFs enables us to find out which RFs are likely to be the best indicators for age-17 distress (M1). This information seemed relevant, both from a clinical as well as a scientific point of view, to determine which RFs may potentially be most fruitful for settings in which it is not possible to assess and/or utilize as many as 10 RFs. Secondly, we aimed to find out whether the same RFs are the best indicators for age-17 distress, when age-14 distress is taken into account (M1 vs M3). RFs that have a high relative importance in explaining age-17 distress, even after taking age-14 distress into account, are more likely to have a unique effect in the development of distress. Moreover, we repeated the analyses separately for the CA+ and the CA- group as well as for males and females, to investigate differences in result patterns between subgroups.

Third, we performed a series of prediction analyses to test with what accuracy the RFs (and/or distress) at age 14 predict distress at age 17. More specifically, in contrast to the above described analyses that focussed on the explanatory value of the RFs (and/or distress) at age 14, the prediction analyses had the aim to find out how well RFs (and/or distress) at age 14 can forecast the level of distress at age 17, which then can be compared back to the actual observed distress level at age 17 and thereby provides information on the predictive accuracy. We again used the three main models described above (M1, M2 and M3). Here, we again conducted the three main models for two reasons. Firstly, we aimed to investigate whether RFs (M1) have a similar predictive accuracy as age-14 distress (M2). This comparison seemed important, as the predictive accuracy of previous distress on future distress has been investigated, but very little is known about the predictive accuracy of RFs. Secondly, we aimed to find out whether the combination of RFs and age-14 distress is better than one information source alone (M3 vs M1 and M2), as this may be important knowledge for future research on risk and/or mental health screening. All three prediction models were conducted once as a *categorical* model, with general distress at age 17 as categorical outcome variable, and once as *linear* model, with general distress at age 17 as a continuous outcome variable. For the categorical distress variable we conducted a series of factor mixture models (Clark et al., 2013), which are hybrid models that add latent classes on top of the latent factors, with different invariance levels between the classes. We did this to classify the adolescents based on their distress profiles into categorical distress classes, while also taking into account the continuous nature of distress. Firstly, we applied latent class analyses to identify possible class solutions and then conducted one-factor mixture models with the appropriate class solutions (factor mixture model analyses details can be found in Appendix D.2). For a factor mixture model solution with two classes we planned to use *logistic* prediction models, for a factor mixture model solution with three or more unordered classes we planned to use *multinomial* prediction models, and

for a factor mixture model solution with three or more ordered classes we planned to use *ordinal* prediction models.

A common problem in prediction-based modelling is “overfitting”. The problem of overfitting is that a model describes the data of the (sub-)sample at hand very well, but is so strongly calibrated to the data at hand that it does not generalize well to other (sub-)samples and therefore only reaches a low predictive accuracy (Huys, Maia, & Frank, 2016). In data-driven prediction frameworks three analytical procedures are often, and sometimes in combination, used to reduce the risk of overfitting. One approach is dimensionality reduction, which reduces the number of variables to reach a more appropriate number of variables in proportion to the number of observations (e.g. via principal component or factor analysis; Huys et al., 2016). A second approach is cross-validation, which divides the data into a “training” sub-set and a left-out “testing” sub-set. This enables the researcher to estimate and/or calibrate the model in the “training” data sub-set, and to subsequently test the predictive accuracy of the developed model in the left-out “testing” data sub-set (e.g. via random or quasi-random sample splitting; Huys et al., 2016; Yarkoni & Westfall, 2017). Thus, “reduction in overfitting – obtained by ensuring that no single data point is used in both the training and evaluation of a model” (p. 12; Yarkoni & Westfall, 2017). However, strictly speaking “cross-validation provides a means of estimating how capably a model can generalize to new data . . . [but] does not directly prevent overfitting” (p. 13; Yarkoni & Westfall, 2017). A third approach is regularization, which can include variable selection, as in setting coefficients of variables that have small effects to zero and thereby excluding them from the analysis, and/or variable penalization, which does not necessarily exclude variables, but shrinks their regression coefficients (Huys et al., 2016). For example, the aim of lasso regularization is to find “an optimal compromise (optimal in the sense of minimizing the cost function) between the two competing goals of (a) minimizing the sum-of-squares and (b) having as small an absolute sum [of the regression coefficients] as possible” (p. 13; Yarkoni & Westfall, 2017). This should ideally reduce the risk that “available predictors will happen to capture some variation in the training sample observations purely by chance” (p. 15; Yarkoni & Westfall, 2017).

To reduce the risk of “overfitting”, I strived to take into account all three approaches. However, it is important to emphasise that my modelling approach was predominantly theory-driven and not data-driven. That is, all included RFs were derived from a previously conducted systematic review (see Chapter 2). Therefore, I adopted some of the three approaches indirectly. The first consideration, dimensionality reduction, was already implemented a priori, as all of the 10 RFs were derived from our systematic review (see Chapter 2) and were factor analysed (of note, expressive suppression was not factor analysed as this was measured with only one item). For details regarding the factor analyses see the above paragraph on “variable estimation” and/or Appendix D.1. The second consideration, cross-validation, was implemented in the classical sense. More specifically, we split the sample quasi-randomly into a training sample (75%; n = ~850) and a testing sample (25%; n = ~280; quasi-randomly means that that the relative class proportion of age-17 distress was kept equal between the training and the testing sample). We chose to have a larger training than testing sample, to be able to estimate as accurate prediction models as possible, particularly given that categorical prediction models require a substantial amount of power (relatively more than linear models, depending on the category number

and size of the outcome variable). The third consideration, regularization, was adopted indirectly. We decided not to penalize coefficients in a data-driven way, due to three reasons. Firstly, according to Williams (2020; p. 20) the "predictive advantage of regularization depends on the signal-to-noise ratio . . . [and] dissipates with more signal and when p / n → 0", with "p" being the number of variables and "n" the number of observations, which in our case would have been notably small (e.g. [10 RFs + age-14 distress + CA + gender] / 1130 participants = 0.01). Secondly, regularization techniques have not yet been developed and implemented for all types of categorical prediction models, making it practically impossible to pre-plan the adoption of such penalization methods for our modelling procedure (for details see Kuhn, 2018). Thirdly, and most importantly, penalizing variables via regularization is a rather data-driven approach and our modelling framework was predominantly theory-driven, which is why we decided to adopt variable selection in a more theoretically informed approach within a set of post-hoc exploratory analyses. Those analyses are detailed below in the paragraph "exploratory post-hoc analyses". We did however calibrate our categorical prediction models with regard to the best link function (i.e. logistic or probit), using the Akaike Information Criterion (AIC) and the residual deviance as model comparison indices.

We then used the models resulting from the training procedures (n = ~850) to predict distress at age 17 in the testing sample (n = ~280). To evaluate *categorical* prediction models, we calculated the amount of predicted distress scores that were predicted into their observed distress class. To evaluate the *linear* prediction models, we used the standard errors (SEs) of the age-17 distress factor scores and computed person-specific 95% confidence intervals (CI). We then calculated for how many adolescents our model could predict distress scores that fell into their respective 95% factor score CI. We again, also computed the analyses separately for the CA+ and the CA- group as well as for males and females, to investigate differences in result patterns between subgroups. This time, we could quantify the differences between the CA and the gender subgroups using proportion comparison tests, as we could describe the determined accuracies as accuracy proportions.

**Exploratory post-hoc analyses.** As one of the motivations for conducting classical prediction models was to aid translational research on risk and mental health screening, we additionally explored whether a selected sub-set of predictor variables would provide similar information as all 10 RFs, CA and gender (i.e. M1). We deemed those exploratory analyses as important, as such an assessment may be more feasible and efficient in many non-clinical settings that lend themselves well for risk and mental health screening (e.g. school settings). To this end, we re-ran all prediction models this time only including a selected subset of those RF predictors that had the highest relative importance and were (at least marginally (<0.10)) significant in the majority of the subgroups (i.e. in the CA+, CA-, female and male sub-groups). Hence, the post-hoc analyses are decidedly exploratory and need to be interpreted with caution, but I believe that they are very informative for translational screening research.

**Software.** Most analyses were performed in R version 3.5.1 (R Core Team, 2018; R packages are reported in Appendix D.3). The factor scores and SEs for age-14 and age-17 distress were estimated in MPlus 8.2 (Muthén & Muthén, 2017), as it was not possible to compute the SEs based on categorical data in R. Similarly, we performed the latent class and factor mixture model analyses in MPlus as this allowed us to specify the items as categorical (Muthén & Muthén, 2017).

**Data availability.** Data for this specific paper has been uploaded to the Cambridge Data Repository https://doi.org/10.17863/CAM.46642 and is password protected. Our participants did not give informed consent for their measures to be made publicly available, and it is possible that they could be identified from this data set. Access to the data supporting the analyses presented in this paper will be made available to researchers with a reasonable request to openNSPN@medschl.cam.ac.uk.

**Code availability.** Analysis code is available on https://osf.io/rnsdj/.

## 5.3 RESULTS

### 5.3.1 Sample

As none of the adolescents qualified as outlier in the multivariate space, we could include 1130 adolescents of which 631 were exposed (CA+) and 499 were not exposed to prior CA (CA-; see Table 5.1). The CA groups did not differ in age or gender proportions. SES was higher and a prior psychiatric history was less likely in the CA- than in the CA+ group. Of the 1130 participants, 620 were female and 510 male. The male and the female groups did neither differ in age nor SES. Female adolescents were more likely to have a prior psychiatric history.

Table 5.1
*Sample description, split for CA and gender*

| | **CA+ (n = 631)** | **CA- (n = 499)** | **$\chi^2$ /z / t** | **p-value** |
|---|---|---|---|---|
| gender | Female = 358<br>Male = 273 | Female = 262<br>Male = 237 | 1.85 (1) | .17 |
| age 14* | 14.49 (0.28) | 14.48 (0.28) | -0.43 (1049.3) | .67 |
| age 17* | 17.49 (0.34) | 17.48 (0.32) | -0.48 (1015.8) | .63 |
| SES | Hard pressed = 73<br>Moderate means = 36<br>Comfortably off = 168<br>Urban prosperity = 37<br>Wealthy achievers = 317 | Hard pressed = 30<br>Moderate means = 11<br>Comfortably off = 104<br>Urban prosperity = 41<br>Wealthy achievers = 313 | 5.24 | <.001 |
| prior psychiatric history at age 14 | Yes = 199<br>No = 432 | Yes = 74<br>No = 425 | 41.54 (1) | <.001 |
| prior psychiatric history at age 17* | Yes = 267<br>No = 297 | Yes = 122<br>No = 345 | 48.05 (1) | <.001 |
| | **Female (n = 620)** | **Male (n = 510)** | **$\chi^2$ /z / t** | **p-value** |
| age 14* | 14.49 (0.27) | 14.48 (0.29) | 0.61 (1027) | .54 |
| age 17* | 17.50 (0.32) | 17.47 (0.34) | 1.38 (954.71) | .17 |
| SES | Hard pressed = 51<br>Moderate means = 23<br>Comfortably off = 154<br>Urban prosperity = 34<br>Wealthy achievers = 358 | Hard pressed = 52<br>Moderate means = 24<br>Comfortably off = 118<br>Urban prosperity = 44<br>Wealthy achievers = 272 | 1.33 | .18 |
| prior psychiatric history at age 14 | Yes = 176<br>No = 444 | Yes = 97<br>No = 413 | 12.90 (1) | <.001 |
| prior psychiatric history at age 17* | Yes = 249<br>No = 326 | Yes = 140<br>No = 316 | 16.66 (1) | <.001 |

*Note.* For age I depict the mean values and the belonging standard deviations in brackets. The Pearson's $\chi^2$ tests were used for binary data and performed with Yate's continuity correction. The z-test was used for the SES variable and was conducted as asymptotic linear-by-linear association test, to account for the ordering in the data. The t-tests were used for continuous data and were conducted as Welsh's two-sample t-tests. SES was calculated based on the ACORN classification system (http://www.caci.co.uk; Morgan & Chinn, 1983). Prior psychiatric history was measured with the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Present and Lifetime Version; Kaufman et al., 1997) and included learning disabilities, clinical sub-threshold diagnoses and deliberate self-harm at age 14; and clinical sub-threshold diagnoses and deliberate

self-harm, but not learning disabilities, at age 17. *Please note, the descriptive statistics are not based on the imputed data, which is why some participants have missing data on some descriptive variables, and accordingly some numbers do not add up. Tests were conducted two-sided.

### 5.3.2 Disentangling the Amount of Variance that RFs and Age-14 Distress Explain in Age-17 Distress

As described above, we conducted three main models. In addition to CA and gender, the first model contained the ten RFs (model M1), the second model contained age-14 distress (model M2), and the third model contained both the RFs and age-14 distress (model M3) as regressors to explain age-17 distress. Adding the RFs to CA and gender significantly improved the model and increased the explained variance from 4 to 20% (see Likelihood-Ratio test for M1 in Table 5.2). Similarly, adding age-14 distress (instead of the RFs) to CA and gender significantly improved the model and increased the explained variance from 4 to 23% (see Likelihood-Ratio test for M2 in Table 5.2; see Appendix D.4 for Figures depicting change in distress). Adding age-14 distress to the model with CA, gender and the RFs improved the model significantly and increased the explained variance from 20 to 24% (see Likelihood-Ratio test for M3-D14 in Table 5.2). Adding the RFs to the model with CA, gender and age-14 distress increased the explained variance from 23 to 24%, but did not improve the model significantly ($p = 0.07$; see Likelihood-Ratio test for M3-RFs in Table 5.2). Hence, the RFs seemed to explain age-17 distress significantly, but seemed to explain the change in distress from age 14 to age 17 at best marginally. Importantly, there was no multicollinearity between the RFs and age-14 distress (see Appendix D.5). When computing the analyses separately for the CA+ and the CA- group (CA+: M1 = 17%, M2 = 19%, M3 = 21%; CA-: M1 = 22%, M2 = 25%, M3 = 26%), or for females and males (females: M1 = 19%, M2 = 24%, M3 = 26%; males: M1 = 20%, M2 = 20%, M3 = 23%), the result patterns remained similar.

Table 5.2
*Linear regression models*

| | Model | b | p-value | $R^2$ | $R^2$ adj | LRT(df) | p-value |
|---|---|---|---|---|---|---|---|
| *Baseline model I (B1): With CA as regressor* | | | | | | | |
| *B1* | CA | 0.44 | <.001* | 3% | 3% | | |
| *Baseline model II (B2): Adding gender to B1* | | | | | | | |
| | | | | | | *compared against B1* | |
| *B2* | gender | -0.30 | <.001* | 4% | 4% | 25.33(1) | <.001* |
| *Adding RFs and age-14 distress (D14) separately to B2* | | | | | | | |
| | | | | | | *compared against B2* | |
| *M1* | RFs | -0.86 | - | 20% | 19% | 297.25(10) | <.001* |
| | | | | | | *compared against B2* | |
| *M2* | D14 | 0.63 | <.001* | 23% | 23% | 357.07(1) | <.001* |
| *Adding RFs and age-14 distress (D14) together to B2* | | | | | | | |
| | | | | | | *compared against M2* | |
| *M3* | RFs | -0.19 | - | 24% | 23% | 22.11(10) | .07 |
| | | | | | | *compared against M1* | |
| *M3* | D14 | 0.54 | <.001* | 24% | 23% | 81.93(1) | <.001* |

*Note.* adj = adjusted. LRT = Likelihood-Ratio test. There is no p-value for the RFs in model M1 and M3, as the bs of the RFs are here summed up to illustrate whether the cumulative effect is positive or negative, but as the RFs are 10 individuals regressors there is no cumulative p-value.

### 5.3.3 Disentangling the Relative Importance of RFs and Age-14 Distress in Explaining Age-17 Distress

We next decomposed the individual variance contribution of the regressors. The self-esteem and brooding RFs explained most and expressive suppression explained the least amount of variance. Moreover, when taking age-14 distress into account the importance ranking of the RFs stayed the same as in the model without age-14 distress (i.e. compare M1 and M3). In the model including both age-14 distress and the RFs, the RFs explained more variance in age-17 distress than age-14 distress (M3 RFs total variance = 57%; M3 age-14 distress total variance = 37%; see Table 5.3). The result pattern remained comparable when being computed separately for CA+ (M3 RFs total variance = 62%; M3 age-14 distress total variance = 37%) and CA- groups (M3 RFs total variance = 58%; M3 age-14 distress total variance = 39%), as well as for female (M3 RFs total variance = 54%; M3 age-14 distress total variance = 43%) and male participants (M3 RFs total variance = 63%; M3 age-14 distress total variance = 30%).

Table 5.3
*Relative importance analyses for the whole group: for RFs only (M1), age-14 distress only (M2), and RFs and age-14 distress together (M3)*

| Variable | % M1: RFs only | bootstrap CI | % M2: D14 only | bootstrap CI | % M3: RFs & D14 | bootstrap CI |
|---|---|---|---|---|---|---|
| **Abs** | 19.94 | - | 23.10 | - | 24.27 | - |
| CA | 06.74 | 02.03-13.11 | 08.37 | 03.34-15.52 | 05.00 | 01.47-10.05 |
| Gender | 02.30 | 00.58-06.28 | 03.16 | 01.18-07.28 | 01.37 | 00.49-04.08 |
| **Total** | 09.03 | - | 11.53 | - | 06.37 | - |
| Neg. self-esteem | 21.12 | 14.84-28.31 | - | - | 12.58 | 09.80-16.18 |
| Pos. self-esteem | 17.79 | 10.64-24.67 | - | - | 11.25 | 06.64-16.28 |
| Brooding | 16.10 | 09.86-23.02 | - | - | 09.58 | 06.56-13.80 |
| Family cohesion | 08.72 | 04.42-14.17 | - | - | 05.71 | 02.88-09.55 |
| Aggression | 07.03 | 02.30-13.95 | - | - | 04.11 | 01.54-08.93 |
| Friendships | 05.81 | 02.47-10.77 | - | - | 03.60 | 02.06-06.69 |
| Family support | 05.03 | 02.17-10.13 | - | - | 03.44 | 01.50-07.08 |
| Reflection | 04.50 | 02.55-08.10 | - | - | 03.05 | 02.07-05.15 |
| Dis. tolerance | 04.17 | 01.31-08.45 | - | - | 02.79 | 00.98-05.85 |
| Expressive sup. | 00.72 | 00.10-03.62 | - | - | 00.43 | 00.06-02.58 |
| **Total** | 90.97 | - | - | - | 56.55 | - |
| D14 | - | - | 88.48 | 80.44-94.15 | 37.08 | 28.76-43.14 |
| **Total** | - | - | 88.48 | - | 37.08 | - |

*Note.* D14 = age-14 distress, CI = confidence interval, Abs = absolute amount of explained variance, CA = childhood adversity, Neg. = negative, Pos. = positive, Dis. = distress, sup. = suppression.

### 5.3.4 Disentangling the Accuracy with Which RFs and Age-14 Distress Predict Age-17 Distress

We first performed a series of factor mixture models to classify the adolescents based on their categorical distress profiles, while also taking into account the continuous nature of distress. The three-class model, which allows the factor score mean to vary per distress class (called factor mixture model-1; for more specific analysis details see Appendix D.2), performed well (entropy = 0.97) and revealed a theoretically plausible solution, splitting the adolescents into “low/mild”, “moderate” and “high” distress severity classes. Figure 5.1 shows the class solution plotted against the continuous general distress scores. As the best class solution was ordered categorical, we conducted three *ordinal* prediction models with the three-class distress variable as outcome variable. Of the three models one again

contained the RFs (M1), one age-14 distress (M2), and one both (RFs and age-14 distress; M3) in addition to gender and CA as predictors. The applied ordinal regression models have a proportional odds assumption, which was not met for all predictors. Therefore, we conducted the ordinal regressions as partial proportional odds models and relaxed the proportional odds assumption for those predictors that did not meet the assumption (see details in Appendix D.6).

The three models (M1-M3) had a low to acceptable accuracy ranging from 62% to 64% (see Table 5.4). Hence, about 2 out of 3 adolescents were correctly predicted into their distress severity class, regardless of using RFs, age-14 distress, or both as predictors for age-17 distress. Once more, the results were generally comparable when we split the adolescents into CA+ (accuracy: M1 = 54%, M2 = 60%, M3 = 58%), CA- (accuracy: M1 = 66%, M2 = 69%, M3 = 69%), female (accuracy: M1 = 58%, M2 = 59%, M3 = 58%) and male groups (accuracy: M1 = 61%, M2 = 64%, M3 = 61%). More specifically, the prediction accuracy did not differ between the CA and gender subgroups (for details see Appendix D.7); only model M1 revealed a borderline effect for the CA+ vs CA- comparison ($Chi^2 = 3.82$, df = 1, $p = 0.051$).

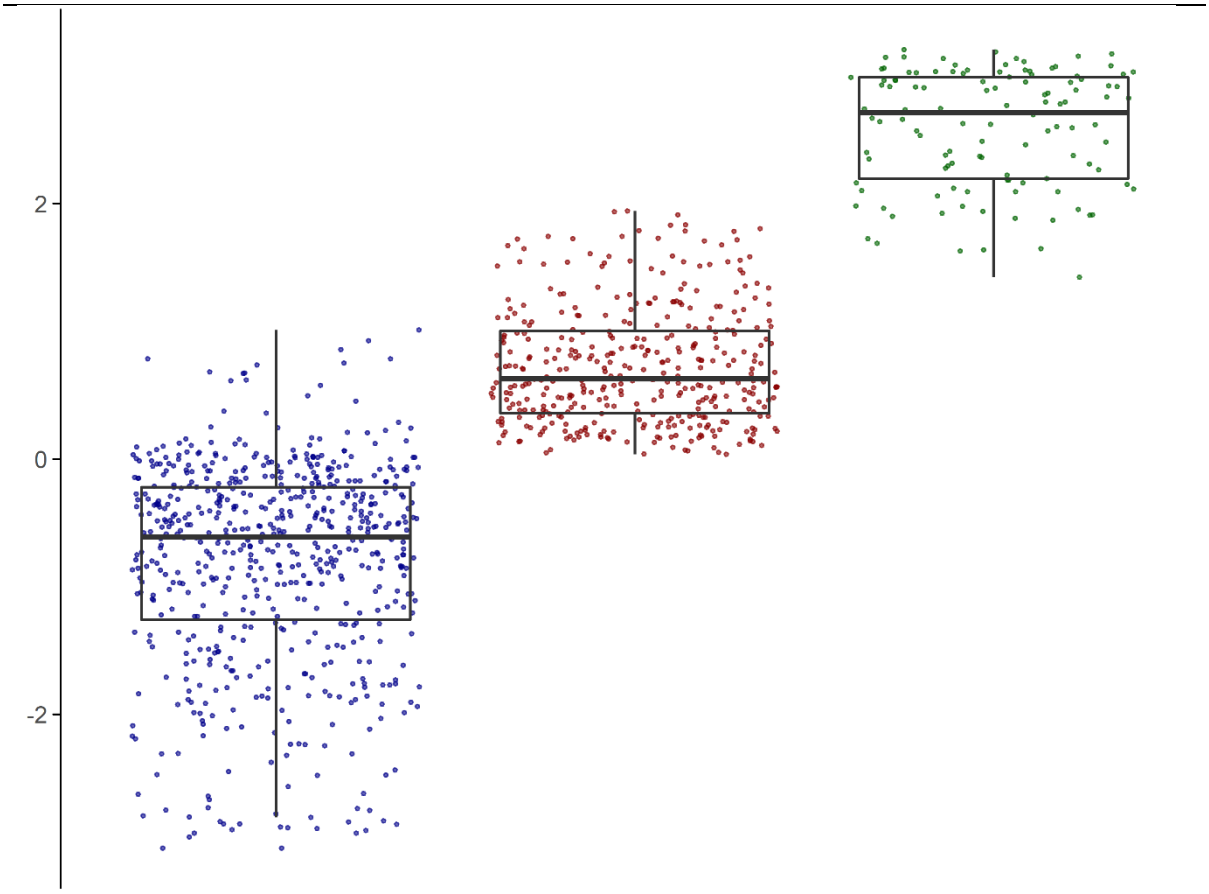



*Figure 5.1.* Three-class distress solution (low/mild: n = 623; moderate: n = 390; high: n = 117) plotted against the continuous distress severity scores. Center line = median (50% quantile); lower box limit =25% quantile; upper box limit = 75% quantile; lower whisker = smallest observation greater than or equal to the lower box limit -1.5 x Inter Quartile Range (IQR); upper whisker = largest observation less than or equal to the upper box limit + 1.5 x Inter Quartile Range (IQR).

Table 5.4
*Ordinal prediction analyses for the whole group: for RFs only (M1), age-14 distress only (M2), and RFs and age-14 distress together (M3)*

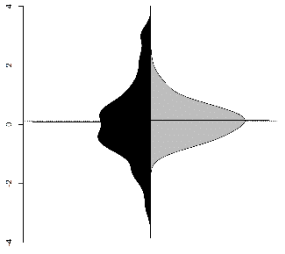
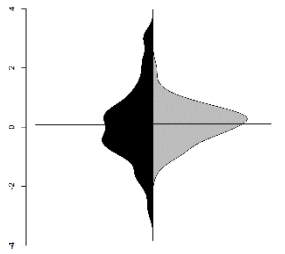
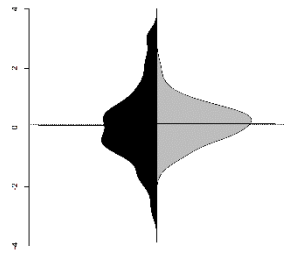
| | **M1: RFs only** | | **M2: D14 only** | | **M3: RFs & D14** | |
|---|---|---|---|---|---|---|
| | observed | predicted | observed | predicted | observed | predicted |
| Residual deviance | 1420.35 | - | 1390.41 | - | 1392.83 | - |
| ROC | - | low=0.70<br>mod=0.65<br>high=0.75 | - | low=0.69<br>mod=0.68<br>high=0.71 | - | low=0.69<br>mod=0.68<br>high=0.74 |
| Sensitivity | - | low=0.83<br>mod=0.54<br>high=0.00 | - | low=0.79<br>mod=0.53<br>high=0.00 | - | low=0.82<br>mod=0.53<br>high=0.00 |
| Specificity | - | low=0.52<br>mod=0.79<br>high=1.00 | - | low=0.50<br>mod=0.76<br>high=1.00 | - | low=0.52<br>mod=0.77<br>high=1.00 |
| Accuracy | - | 0.64<br>low=0.68<br>mod=0.66<br>high=0.50 | - | 0.62<br>low=0.65<br>mod=0.64<br>high=0.50 | - | 0.63<br>low=0.67<br>mod=0.65<br>high=0.50 |
| Low distress severity | 155 | 189 of which<br>- 129 correct<br>- 44 false mod<br>- 16 false high | 155 | 186 of which<br>- 123 correct<br>- 46 false mod<br>- 17 false high | 155 | 187 of which<br>-127 correct<br>- 45 false mod<br>- 15 false high |
| Mod distress severity | 97 | 91 of which<br>- 52 correct<br>- 26 false low<br>- 13 false high | 97 | 95 of which<br>- 51 correct<br>- 32 false low<br>- 12 false high | 97 | 93 of which<br>- 51 correct<br>- 28 false low<br>- 14 false high |
| High distress severity | 29 | 1 of which<br>- 00 correct<br>- 00 false low<br>- 01 false mod | 29 | 0 of which<br>- 00 correct<br>- 00 false low<br>- 00 false mod | 29 | 1 of which<br>- 00 correct<br>- 00 false low<br>- 01 false mod |

*Note.* D14 = age-14 distress. Mod = moderate. All models were computed with childhood adversity and gender as predictors. ROC = receiver operating characteristic. Accuracy = relative number of correctly predicted cases. Sensitivity = e.g. for low distress: the number of adolescents who are correctly predicted into the low distress group divided by all adolescent who are actually in the low distress group. Specificity = e.g. for low distress: the number of adolescents who are correctly not predicted into the low distress group divided by all adolescent who are actually not in the low distress group. Variable for which the proportional odds assumption was relaxed can be found in Appendix D.6.

We next tested the prediction accuracy for *linear* models with the continuous distress severity variable as outcome measure. These analyses revealed that in contrast to the *ordinal* models, the prediction accuracy for all three *linear* models was low (37 to 41%; Table 5.5), as the age-17 distress level of only about two in five adolescents was predicted accurately. Similar findings were revealed when splitting the group based on CA (CA+: M1 = 34.62%, M2 = 32.69%, M3 = 36.54%; CA-: M1 = 40.32%, M2 = 38.71%, M3 = 38.71%) and gender (female: M1 = 32.90%, M2 = 35.53%, M3 = 34.87%; male: M1 = 38.89%, M2 = 41.27%, M3 = 42.06%). Once more, the prediction accuracy did not differ significantly between the CA and gender subgroups (see Appendix D.7).

Table 5.5
*Linear prediction analyses for the whole group: for RFs only (M1), age-14 distress only (M2), and RFs and age-14 distress together (M3)*

| | **M1: RFs only** | | **M2: D14 only** | | **M3: RFs & D14** | |
|---|---|---|---|---|---|---|
| | **observed** | **predicted** | **observed** | **predicted** | **observed** | **predicted** |
| RMSE | 1.18 | 1.18 | 1.15 | 1.13 | 1.16 | 1.13 |
| $R^2$ | 0.18 | 0.16 | 0.23 | 0.22 | 0.22 | 0.22 |
| MAE | 0.90 | 0.91 | 0.87 | 0.88 | 0.88 | 0.88 |
| Accuracy | - | 37.14% | - | 40.00% | - | 40.71% |

| | | | | | | |
|---|---|---|---|---|---|---|
| predicted into $CI_{95\%}$ | - | 104 | - | 112 | - | 114 |
| not predicted into $CI_{95\%}$ | - | 176 | - | 168 | - | 166 |
| Accuracy plots<br>x-axis:<br>observed (left = black) vs predicted (right = grey) distribution<br>y-axis:<br>distress level |  | |  | |  | |

*Note.* D14 = age-14 distress. All models were computed with childhood adversity and gender as predictors. RMSE = root mean squared error, MAE = mean absolute error, Accuracy = relative number of correctly predicted cases. Model accuracy was based on 1000 bootstraps.

### 5.3.5 Post-hoc Exploration: Disentangling the Accuracy for Fewer RFs Predicting Age-17 Distress

Negative self-esteem, positive-self-esteem, and brooding had in three of the four subgroups the highest relative importance. Moreover, those three RFs were (at least marginally (<0.10)) significant in our RF regression models (i.e. the M1s), in three of the four subgroups. Therefore, we next re-ran all prediction models this time instead of including all 10 RFs, CA and gender, only including these three RFs and gender. Interestingly, in these post-hoc analyses, both the ordinal and the linear models performed similar as the models including all RFs (change in accuracy: ordinal models from 64% to 62%, $Chi^2$ = 0.37, df = 1, $p$ = 0.54; linear models from 37.14% to 37.14%, $Chi^2$ = 0, df = 1, $p$ = 1). Moreover, the models including gender, the three RFs and age-14 distress were rather comparable to the models including gender, CA, all 10 RFs, and age-14 distress (i.e. M3; change in accuracy: ordinal models from 63% to 60%, $Chi^2$ = 0.61, df = 1, $p$ = 0.44; linear models from 40.71% to 40.71%, $Chi^2$ = 0, df = 1, $p$ = 1). For completeness, we also conducted the prediction analyses with a subset of the RFs separately in the subgroups, which can be found in Appendix D.8.

## 5.4 DISCUSSION

We aimed to shed light onto potentially promising RF targets that reduce subsequent distress, by pursuing three sub-goals: *First,* we intended to find out to which degree RFs can explain subsequent distress. Our results suggest that RFs explained a similar amount of variance in age-17 distress as age-14 distress could explain, but explained at best a marginal amount of variance beyond the variance explained by age-14 distress. However, when used together RFs had a higher relative importance than age-14 distress for explaining age-17 distress. *Second,* we aimed to find out which RFs are the best indicators for subsequent distress. Our results showed that self-esteem and brooding RFs revealed significance in the multivariable regression models and had the highest relative importance. *Third,* we intended to explore with what accuracy RFs can predict distress levels three years later. We found that RFs and distress at age 14 were similarly accurate in predicting distress at age 17. The prediction accuracy was low and highly unsatisfactory when we predicted continuous distress scores. When we predicted more crude ordinal (“low”, “moderate” and “high”) distress classes the accuracy was again

not good, but acceptable. As such, both RFs and distress at age 14 (as well as their combination) are able to correctly predict the categorical distress class of about 2 in 3 adolescents.

RFs and/or age-14 distress explained about one-fifth to one-fourth of the overall variance in distress three years later. Importantly, this was after CA and gender were taken into account. Hence, despite the fact that we have used gender, life-history information (i.e. CA), a broad range of distress symptoms and as many as 10 empirically supported RFs, we were only able to explain a small proportion of the variance in distress three years later. This is alarming and interesting at the same time. Dinga and colleagues (2018) put forward the explanation that the way psychopathology is defined may lack important information (i.e. content validity), such as biological components, which may make it so difficult to predict it well. Another explanation could be derived from the time period we have investigated. We assessed the adolescents during early (age 14) and later (age 17) adolescence, which is generally described as a particularly malleable period during which a lot of mental health problems develop (World Health Organization, 2018)**.** That is, explaining and predicting distress over a period during which many mental health problems manifest themselves may be particularly difficult. A third account may come from the instructions that were provided for the assessment of the distress symptoms: “please tick how often you have felt or acted in this way over the past two weeks”. The instructions assess distress during the past two weeks, which for some adolescents may have captured state- rather than trait-distress. An outcome construct that at least to some extent captures state characteristics may complicate explanation and prediction even further. In sum, insufficient content validity, a sensitive developmental time period, and state-like characteristics of the distress variable may all help explain why it was so difficult to explain as well as predict subsequent distress.

While the RFs explained age-17 distress significantly, the RFs explained at best a marginal amount of variance beyond the variance explained by age 14-distress. However, RFs not only had a higher relative importance than age-14 distress when both were used together to explain age-17 distress, but the RFs and age-14 distress had a similar accuracy for predicting age-17 distress. This clearly is a notable finding, as RFs could similarly well predict distress over the course of three years, as distress could predict itself over the course of three years. Moreover, a combination of the two information sources (RFs and age-14 distress) did not necessarily seem advantageous above either source alone. Of note, there was no overlap between RF and distress items content-wise, and no multicollinearity between RFs and age-14 distress. Therefore, if our results were to be replicated, I would assume that knowledge on the RFs may, due to its “conceptual commitment to strengths and assets” (see p. 136 in Luthar, Lyman, & Crossman, 2014), be highly interesting for various public health and clinical settings. More specifically, in settings where a strengths-focus would be more feasible than a symptom-focus, RFs could be assessed to screen and monitor mental health.

If I would have to judge which of the RFs may be the most promising for screening and monitoring mental health, I probably would choose brooding and the self-esteem RFs. Those RFs had the strongest relative importance for explaining subsequent distress and were significant in the multivariable RF model (M1). Moreover, the importance ranking of the RFs for explaining age-17 distress did not change when taking age-14 distress into account, which suggests that particularly brooding and self-esteem RFs have unique effects on the development of distress. Critics might

question whether explanatory, amenable RFs are indeed helpful for the development of predictive screens. Even though a high explanatory value of variables does not always result in a high predictive accuracy (and particularly not vice versa), explanatory variables that "successfully capture important aspects of human psychology should be much more likely to survive . . . [predictive] tests" (p. 19; Yarkoni & Westfall, 2017). This was clearly reflected in our findings. Our prediction results remained rather stable when we used only brooding and self-esteem instead of all 10 RFs and/or age-14 distress as predictors. Specifically, brooding and self-esteem RFs seemed to forecast subsequent distress (62% accuracy) equally well as distress could forecast itself (62% accuracy).

Assuming that our findings are correct and that RFs and mental distress indeed forecast subsequent distress equally well, critics might further question whether the usage of RF screens can have practical advantages above and beyond distress-focussed screens. I do not believe that we have a definite answer to this question, but I will discuss some potential advantages of RF screens. First of all, the brooding and self-esteem RFs were together measured with only 15 items, which is much less than the 41 distress items and suggests that an RF screen may be more time-efficient. Second, although knowledge on the effects of RFs is more limited than knowledge on the effects of general distress, the finding that both self-esteem and brooding seem to play an important role in the development of mental health problems has been noted in various previous research, and the suggestion to use self-esteem (Keane & Loades, 2017) or brooding (Young & Dietrich, 2014) as time-efficient mental health screens is not without precedent. Young and Dietrich (2014) for example employed the same brooding subscale as used in our study (5 items of the RRS; Treynor et al., 2003) and detected a screening accuracy of 91 percent for concurrent depressive symptoms in young adolescents. Third, an RF-focus, as opposed to a symptom-focus, has been suggested to be less stigma-prone (Bolier et al., 2013), to potentially reduce the risk of inducing negative feelings and to be more acceptable. This conjecture clearly needs formal testing. However, if it would hold, it would come with highly crucial information for stigma-sensitive and risk-prone screening settings. A more detailed exploration of the viability of using RFs as screening tools is out of the scope of this discussion, but the reader can find a more detailed evaluation of this topic, based on the screening criteria put forward by the National Screening Board of Public Health England (Public Health England, 2015), in Chapter 8 (paragraph 8.7.1; "How Can RFs Inform Risk Detection and Mental Health Screening?"). Overall, it is important to note that our results require replication in an independent sample, need to be tested in translational studies, would ideally need replication in other populations to ensure a clear scope for generalization, and we would need to succeed in increasing the screening accuracy before any further screening recommendations are warranted. Moreover, an important general limitation is that risk and mental health screening is likely to only be fully beneficial if (a) young people who screen positive but are below-threshold cases for mental disorders can access evidence-based prevention programs, and if (b) young people who screen positive and indeed qualify for a diagnosable mental disorder can access evidence-based treatment programs. This necessity obviously relates to both RF-focussed as well as distress-focussed screening tools in equal measure.

It is important to note that our *linear* prediction models, which are derived from the group level, are not good enough to predict individual-level distress scores three years later. Those models

translated for only two in five adolescents correctly to the individual level. Our *categorical* prediction models, which are also derived from the group level, did predict individual-level distress severity classes better, but there is still plenty of room for improvement. Those models translated for about two-third of the adolescents correctly to the individual level. Hence, the generalization from group to individual level is limited, particularly when predicting *continuous* transdiagnostic distress severity. Therefore, it is crucial that future research identifies ways to increase the prediction accuracy for subsequent distress severity. In sum, I recommend that future research (a) examines whether our findings replicate, (b) tests additional RFs that were not measured in our adolescent cohort but are empirically found to reduce subsequent distress, (c) identifies ways which further increase the prediction accuracy (e.g. shorter prediction intervals), (d) explores whether RFs are indeed less stigma-prone and more acceptable than distress screens, and (e) is conducted at the individual rather than (or in addition to) the group level.

Last but not least, our study is not without limitations. First, ROOTS has a slightly higher than average SES and thus may mainly generalize to more wealthy populations (Goodyer et al., 2010). Second, our latent class and factor mixture model analyses were based on a grandmedian imputation data set, rather than being conducted separately on the 10 imputation data sets, as there is no method and consensus yet on how to pool over class solutions (for details see Appendix D.2). Third, the binary CA variable may not be ideal as it omits the type of the adversity experience, as well as its severity and frequency. Particularly CA severity may be a valuable consideration and addition in future research (Schlechter et al., 2019). However, justification for using CA as a binary indicator stems from research showing that CAs are likely to co-occur and that clustered CA indices have a robust, negative effect on mental health problems (Dunn et al., 2011; Kessler et al., 2010; Schlechter et al., 2019). For future research it would be ideal if adversity would also be assessed, and controlled, for the interim period between the assessment of the RFs and the assessment of subsequent mental distress. Fourth, the RFs were not all assessed with measures developed to particularly reflect the RF construct at hand (e.g. aggression or expressive suppression). Hence, future research should aim to replicate our results with scales particularly developed for the specific RFs, to increase the content validity. Fifth, we only tested 10 RFs, as only those were assessed in our adolescent cohort. However, in the realm of complexity I think that it would be advantageous if future research could assess and test more than 10 empirically-supported RFs. Sixth, our distress index was mainly defined by internalizing (and not externalizing) symptoms and does not contain information on the distress chronicity. Seventh, we built the prediction models on a subset of the ROOTS cohort (n ~850) to predict distress three years later for another ROOTS subset (n ~280). This means that we used data from the same cohort for training and testing our model. However, it may be that adolescents in our cohort are more comparable to each other than to the general population. This would mean that our prediction accuracy would be lower when using our model to predict distress scores for adolescents who did not take part in ROOTS. Therefore, replication of our findings in a different sample is crucial. Eighth, here we mainly focussed on the overall sample and not so much on findings within the subgroups (CA+ vs. CA-, females vs. males). Yet, there were slight differences in the relative importance of the RFs between the subgroups. Future research should more specifically focus on those differences, for example with moderation analyses.

One could argue that investigating age-17 distress as both a categorical and a continuous outcome is superfluous. Yet, I believe that there are good reasons from a scientific as well as a clinical point of view that justify the usage of both (categorical and continuous outcomes) in conjunction. From a statistical point of view it may perhaps seem neater to investigate distress continua. But, first of all our distress classes did take the distress continuum into account, and more importantly, as prior research often only looked at categorical outcomes I feel that it is high time to gain information on the comparison of precise continuous versus more crude categorical outcomes. As our findings showed, it seems like we are not good enough yet to predict precise distress continua, but we are getting into an acceptable range for predicting crude distress classes (from either RFs, distress, or their combination). From a translational point of view, one may favour a categorical outcome as this is often used in clinics, such as cut-offs like "low risk", "at risk/sub-threshold", and "diagnosed". Although crude categorical outcomes may be more easily translatable, providing results of both approaches has given rise to the clinically relevant finding that RFs and prior distress may be promising targets for screens aiming at predicting rough distress risk-categories (e.g. “low”, “moderate”, “high”), but not yet for screens aiming at predicting precise distress risk levels.

As pointed out in the introduction, there is a sparse but ongoing discourse about whether resilience and risk factors are opposing sides of the same coin, which cannot fully be done justice within the scope of this chapter. However, I suggest that future studies could conduct more idiographic rather than group level research, as the “relationship between resilience and risk factors is likely to additionally depend on biological predispositions, type(s) of adversity experienced, the specific environmental circumstances, and the developmental stage” of the adolescent (see Appendix C.16). Moreover, while this chapter specifically focusses on using RFs that explain and predict mental health problems (in individuals with and without CA exposure), it would be interesting to see future research taking the same modelling approach but focussing on those factors that explain and predict a resilient functioning outcome. To this end one could for example focus on explanatory resilience factors reviewed by Kalisch and colleagues (2017; including hair cortisol concentration, trait self-enhancement, expression of specific gene networks, and cortisol stress reactivity), on factors that explain resilient growth trajectories and resilient functioning outcomes as reviewed by Bonanno, Westphal, and Mancini (2011; including perceived control, high positive affectivity, low negative affectivity, trait resilience, low brooding, coping self-efficacy, emotional support, social support, instrumental support, favorable worldviews, and positive emotions), or on factors that relate to resilient functioning specifically following childhood maltreatment, as reviewed in Ioannidis, Askelund, Kievit, and van Harmelen (2020; including the social environment as well as biological factors related to the hypothalamic-pituitary-adrenal axis and polygenetics).

Overall, our results showed that the RFs were able to correctly predict the categorical (“low”/”moderate”/”high”) distress class of 2 in 3 adolescents three years later. This finding was highly similar when predicting age-17 from age-14 distress. The three RFs that were most promising in reducing and predicting subsequent distress were positive self-esteem, negative self-esteem and brooding. Hence, those three RFs may potentially be promising targets for risk-detection and mental health screening, if they hold up in replication and translational research.

Building forth on this knowledge, in the next chapter I aim to shed light on *how* RFs reduce subsequent mental health problems after CA. To this end, I shall examine whether most RFs function as a mediator, as a moderator, or as both for the relationship between CA and subsequent mental health problems.

# CHAPTER 6

# An Empirical Examination of How Resilience Factors Mitigate the Effect of Childhood Adversity on Adolescent Mental Health

Childhood adversity (CA) is often assumed to be a rare incidence (Bonanno et al., 2011). However, as discussed in the previous Chapters, more than one in three people worldwide are exposed to some form of adversity prior to the age of 18 (Kessler et al., 2010). Several large-scale epidemiological studies even reported ratios as high as one in two people (Greif Green et al., 2010; McLaughlin et al., 2012). Generally, CA refers to a single traumatic event (e.g. death of a significant other), or to several severely stressful events (e.g. parental mental illness with significant impact on the family life). According to the CA definition put forward by Katie McLaughlin (2016), CA either comprises a notable divergence of a reasonable living situation, or a notable alteration of the anticipated living situation, and thus demands active adjustment by the child or adolescent (McLaughlin, 2016). Therefore, CA is likely to impact social, emotional, behavioural and cognitive functioning and development (McLaughlin, 2016). The justification of treating CA as an aggregated index, rather than as separate forms of traumatic events or severe stress, stems from a solid body of research showing that different forms of CA often co-occur and that most forms are strongly associated with subsequent mental health problems (Greif Green et al., 2010; Kessler et al., 2010; McLaughlin, 2016; McLaughlin et al., 2012). CA has been found to increase the risk of subsequent mental health problems particularly proximally after the exposure, but it is also found to have a lasting impact across the life span (Greif Green et al., 2010; Kessler et al., 2010). Therefore, it is crucial that we better understand those factors that alleviate the risk and development of mental health problems following CA, so called resilience factors (RFs; see e.g. Chapter 2 or Fritz, de Graaff, et al., 2018).

Simply put, RFs are factors that improve or stabilize mental health in the aftermath of adverse experiences. In our preregistered systematic review, in Chapter 2, my colleagues and I therefore defined RFs as those factors that moderate and/or mediate the relationship between CA and subsequent mental health problems. Importantly, we only included those RFs that function on a social, emotional, cognitive or behavioural level, to ensure that RFs are theoretically amenable to intervention. Here, we investigate three inter-individual (friendship support, family support, family cohesion) and seven intra-individual RFs (positive self-esteem, negative self-esteem, brooding, reflection, distress tolerance, aggression potential, and expressive suppression) that were empirically supported in our systematic review and assessed in our adolescent cohort (Goodyer et al., 2010). Previous research on those 10 RFs has shown that the RFs function as a protective system in which RFs enhance each other less in adolescents with CA than in adolescents without CA, see for example Chapter 3 (or Fritz, Fried, et al., 2018) and Chapter 4 (or Fritz et al., 2019). Moreover, in Chapter 5 (or Fritz, Stochl, Goodyer, van Harmelen, & Wilkinson, 2020), we showed that most of the 10 RFs were not only concurrently associated with less mental health problems, but also three years later. More specifically, the RFs predicted mental distress three years later similarly well as mental distress could predict itself. Hence, there is good support showing that most of the 10 RFs do indeed reduce distress; or in other words,

that RFs have a direct, negative effect on mental health problems. However, as of yet, it is unclear how RFs decrease the detrimental effect of CA on mental health. Therefore, we here aim to investigate whether RFs function predominantly as moderators or as mediators for the positive relationship between CA and subsequent mental health problems. This is important, as moderating and mediating RFs differ theoretically, statistically, and with regard to the inferences that can be drawn (Chmura Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001; Holmbeck, 1997).

*Theoretically*, moderating RFs are RFs that modify the way CA is associated with subsequent mental health problems. That is, the RF operates on the relationship between CA and mental health as it reduces mental health problems more in adolescents with (CA+) than in adolescents without CA (CA-). Taking self-esteem as example, self-esteem would need to reduce mental health problems more in CA+ than in CA- adolescents. Or to put it differently, the negative effect of CA on mental health problems would need to be stronger for adolescents with a low level of self-esteem than for adolescents with a high level of self-esteem. This is depicted in Figure 6.1.a1. For such an effect, CA and the RF should be assessed before the mental health outcome; CA, however, does not necessarily need to have happened prior in time to the RF assessment. Rose, Holmbeck, Millstein Coakley, and Franks (2004) refer to such RFs as “protective factors”. For mediation effects, a timeline is crucial. Here the RF is not

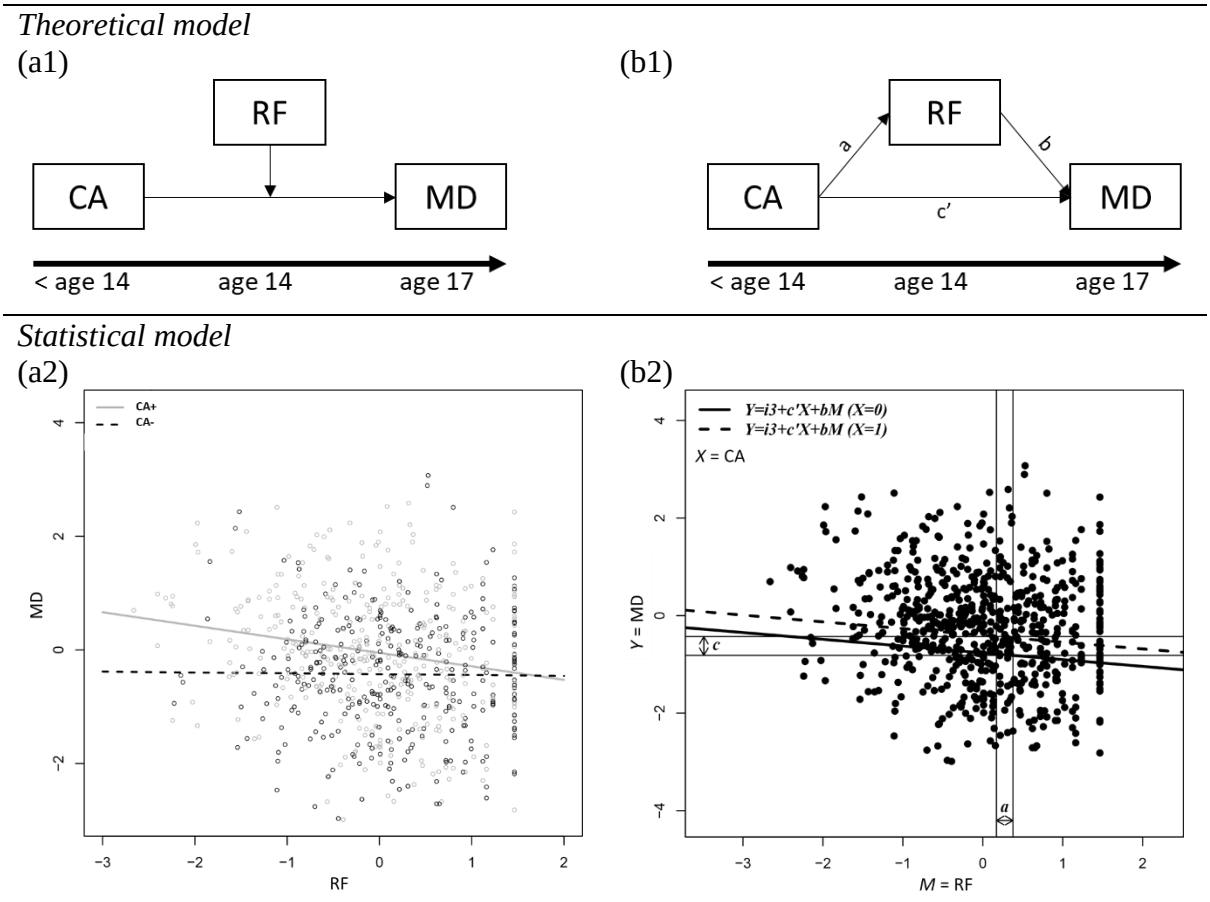



*Figure 6.1.* (a1) = theoretical model for a single moderation effect, (b1) = theoretical model for a single mediation effect, (a2) = statistical model for a single moderation effect, (b2) = statistical model for a single mediation effect. CA = childhood adversity, RF = resilience factors, MD = mental distress/ mental health problems. Model (b1): the a-path represents the effect of CA on the RF; the b-path represents the effect of RFs on MD, while taking the effect of CA on MD into account; the c’-path represents the effect of CA on MD, while taking the effect of RFs on MD into account; the c-path, which is not directly depicted, would represent the total effect of CA on MD, without taking the

effect of RFs into account; the a*b effect, which is not directly depicted, would represent the indirect effect of CA on MD, via the RF. Model (b2): the letters represent the same paths/effects as in the theoretical model in (b1); the dashed regression line depicts the line for adolescents with CA (CA+) and the solid regression line the line for adolescents without CA (CA-); c denotes the total effect CA poses on MD, with the difference representing the mean level difference in MD for CA+ (upper line) and CA- (lower line) adolescents (taking into account the stable intercept); a denotes the total effect CA poses on RFs, with the difference representing the mean level difference in RFs for CA+ (left line) and CA- (right line) adolescents (taking into account the stable intercept); b is represented by the slope of the CA+ as well as the CA- regression line; c' is represented by the distance between the solid CA- regression line and the horizontal line for CA+ adolescents on MD (upper line) located on the vertical CA+ line for RFs (left vertical line); a*b is represented by the distance between the horizontal line for CA- adolescents on MD (lower line) and the solid CA- regression line, located on the vertical CA+ line for RFs (left vertical line). Information regarding the analysis code with which the figure in panel (b2) was create can be found in Appendix E.1.

meant to directly modify the relationship between CA and mental health problems, but is supposed to be the intermediate factor explaining this relationship (at least in part). That is, CA is expected to lead to a lower level of the RF, while a higher level of the RF is expected to lead to less subsequent mental health problems. As mediating RFs are expected to function as intermediate factors, the RF should timewise be assessed in the aftermath of CA, but before the mental health problems. Taking again self-esteem as example, CA is expected to lead to lower self-esteem, while higher self-esteem is expected to lead to less subsequent mental health problems. This is depicted in Figure 6.1.b1. Rose and colleagues (2004) refer to such RFs as "resource factors".

*Statistically,* a moderating RF is supposed to reduce subsequent mental health problems more in CA+ than in CA- adolescents. This is reflected in the regression slope of the RF, which should be significantly steeper in the CA+ than the CA- group. Such an effect is also called an interaction effect and is visualized in Figure 6.1.a2. In contrast, a mediating RF can reduce distress equally in the CA+ and CA- adolescents, as long as the CA exposure results in a lower level of the RF, and a higher level of the RF results in a lower level of mental health problems. Therefore, the regression lines of the RF do not need to differ in steepness between the two groups; but, the regression slope in the CA+ group should reflect a higher level of mental health problems than in the CA- group, at the same level of the RF. This is also called an indirect effect and is visualized in Figure 6.1.b2. Importantly, an RF which reduces mental health problems more in the CA+ than in the CA- group can at the same time be a moderator and a mediator (provided that CA increases subsequent mental health problems, CA is negatively associated with the succeeding RF, and the RF reduces subsequent mental health problems when the effect of CA is taken into account).

Regarding *inferences,* moderating RFs can be considered as mainly advantageous for CA+ adolescents as they reduce distress more in CA+ than CA- group. Mediating RFs can be considered as intermediate, alleviating effect on the pathway from CA to subsequent mental health problems. In this sense, increasing the level of the RF can lead to improved mental health particularly in adolescents with CA (moderator) and/or break or disrupt the chain between CA and mental health problems (mediator). Therefore, both moderating and mediating RFs can serve as prevention and treatment targets for people who have been exposed to CA. Moreover, some RFs reduce subsequent distress significantly for both CA+ and CA- adolescents. A notable advantage of those RFs is that they can serve as prevention or treatment targets for the entire population; for instance, when it is inappropriate or impossible to assess an adversity history or when adolescents with and without CA need to be treated

equally. Overall, I believe that shedding light on effects that describe whether and how RFs reduce mental health problems after CA not only advances empirical knowledge for mental health promotion (Chmura Kraemer et al., 2001; Rose et al., 2004), and theoretical understanding of protective effects in the face of adversity (Holmbeck, 1997; Rose et al., 2004), but could also directly inform translational efforts aimed at reducing mental health problems (Chmura Kraemer et al., 2001; Rose et al., 2004).

In sum, besides exploring direct effects of RFs on subsequent mental health problems, we shall shed light on the question which RFs function as moderator and/or as mediator for the relationship between CA and subsequent mental health problems. We shall investigate this in two ways, firstly by examining single RFs, i.e. "*single* RF models" (see Figure 6.1), and secondly by examining the RFs while taking the effects of the remaining RFs into account, i.e. "*multiple* RF models" (see Figure 6.2). Investigating *multiple* RF models is important, to safeguard overestimating *single* RF effects and to enhance ecological validity.

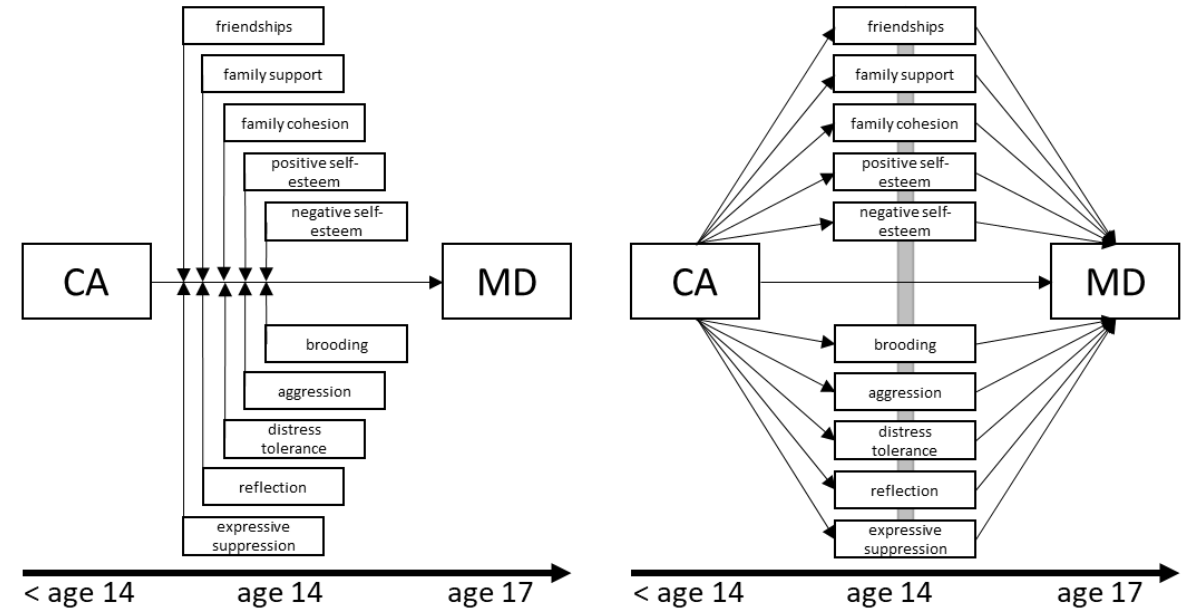



*Figure 6.2.* The left panel depicts a multiple moderator model and the right panel the multiple mediator model, both including all RFs. RFs = resilience factors, CA = childhood adversity, MD = mental distress/ mental health problems. The grey bar in the mediation model indicates that the RFs are allowed to correlate with each other.

## 6.2 METHODS

### 6.2.1 Design

In 2005 and 2006, 1238 adolescents were recruited for the ROOTS study, in schools in and around Cambridgeshire (UK; Goodyer et al., 2010). For inclusion the adolescents had to be 14 years old. The adolescents and their main caregiver provided written informed consent. The adolescents were followed up at age 17 (Goodyer et al., 2010). ROOTS was conducted along the lines of the Declaration of Helsinki, in accordance with Good Clinical Practice guidelines, and was approved by the Cambridgeshire Research Ethics Committee (03/302; Dunn et al., 2011).

### 6.2.2 Sample

Here we include 1130 adolescents for whom we have complete data for CA before age 14, and could impute data for age-14 RFs, and distress at both age 14 and 17.

### 6.2.3 Childhood Adversity (CA)

Adversity before the age of 14 was assessed with the semi-structured Cambridge Early Experiences Interview (CAMEEI), and was performed with the primary caregiver (Dunn et al., 2011; Goodyer et al., 2010). The CAMEEI assessed several different types of CA: family discord, separation for more than six months, living in foster care, lack of maternal affection, lack of maternal engagement, inappropriate parenting, death of a significant other, parental criminality, violence within the family, significant social difficulties within the family, significant and/or chronic mental or physical illness of a parent or sibling, significant external disturbances (e.g. effects of war), significant environmental disturbances (e.g. fire), financial difficulties (with and without parental unemployment), and emotional, physical and/or sexual abuse. Dunn and colleagues (2011) conducted a latent class analysis and clustered the adolescents into no CA, aberrant parenting CA, moderate CA and severe CA. The clustering was performed for the time-windows from zero to five, five to eleven, and eleven to fourteen years, which were used during the assessment of the CAMEEI to enhance recall. Consistent with the previous chapters we clustered adolescents into CA-, which is no CA for all three time-windows, and CA+, which is either of the three types of CA for at least one of the three time-windows. A detailed exploration of the CA subtypes can be found in Dunn and colleagues' (2011) report.

### 6.2.4 Mental Distress

A latent distress factor was estimated based on 13 depression related items, measured with the Short Mood and Feelings Questionnaire (Wood, Kroll, Moore, & Harrington, 1995), and 28 anxiety related items, measured with the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978). Higher scores on the latent distress factor indicate higher levels of internalizing-related mental health problems. In adolescent cohorts, latent distress factors have been found to replicate soundly (Brodbeck et al., 2011; St Clair et al., 2017; Stochl et al., 2015).

### 6.2.5 Resilience Factors

Ten RFs were assessed with questionnaires at age 14. All those RFs were identified by our preregistered systematic review (see Chapter 2) and are amenable, so that the focus lies exclusively on those RFs that are empirically supported and can theoretically, directly be targeted in psychological interventions. The first eight RFs are assessed via adolescent report, the last two via caregiver report:

1. High levels of high friendship support are considered protective and were assessed with five items of the Cambridge Friendships Questionnaire (Goodyer et al., 1989).
2. High levels of high family support are considered protective and were assessed with five items of the McMaster Family Assessment Device (Epstein et al., 1983).
3. High levels of high family cohesion are considered protective and were assessed with the remaining seven items of the McMaster Family Assessment Device (Epstein et al., 1983).

4. High levels of low negative self-esteem are considered protective and were assessed with five items of the Rosenberg self-esteem scale (required reversion of items; Rosenberg, 1965).
5. High levels of high positive self-esteem are considered protective and were assessed with the remaining five items of the Rosenberg self-esteem scale (Rosenberg, 1965).
6. High levels of low ruminative brooding are considered protective and were assessed with five items of the Ruminative Response Scale (RRS; required reversion of items; Burwell & Shirk, 2007; Treynor et al., 2003).
7. High levels of low reflective rumination are considered protective and were assessed with five other items of the RRS (required reversion of items; Burwell & Shirk, 2007; Treynor et al., 2003).
8. High levels of high distress tolerance are considered protective and were assessed with five items of the Emotionality Activity Sociability Temperament Survey (Bould et al., 2013).
9. High levels of low aggression are considered protective and were assessed with four items of the Behaviour Checklist (11 questions based on the DSM-IV criteria for conduct problems; required reversion of items; American Psychiatric Association, 2000; Goodyer et al., 2011).
10. High levels of low expressive suppression are considered protective and were assessed with one item of the Antisocial Process Screening Device (required item reversion; Poythress et al., 2006).

More detailed information regarding the exact content of the RF constructs can be found in Chapter 3. Psychometric information for the RF measures can be found in Appendix B.2.

### 6.2.6 Analysis

Missing data patterns on the RFs and the distress items could be explained by several demographic and life-history variables, such as gender, a CA history, and a prior psychiatric history (for details see Appendix C.4). Therefore, we decided to impute missing data for all RF (103 items) and distress items (122 in total; 41 items + 20 additional items of the full scale for depression related symptoms, for both ages, to include as much information as possible). We used the aforementioned demographic and life-history variables, as well as age and socio-economic status (for details see Table 6.1), as additional auxiliary variables (7 items). We decided not to impute data for the CA variable, as some types of CA did not seem sufficiently predictable (e.g. a fire in the home or a traumatic car crash). Ordinal items were imputed with predictive mean matching and binary items via logistic regression, using multivariate multiple imputation algorithms with chained equations (using 100 iterations; van Buuren & Groothuis-Oudshoorn, 2011). We excluded participants who had more than 85% missing items. Eventually, we could compute 10 complete data sets for 1188 participants. Based on this data we computed the RFs using categorical, confirmatory factor analyses, with a weighted least squares mean and variance corrected (WLSMV) estimator. Similarly, we conducted a strongly invariant categorical confirmatory factor analysis, using the WLSMV estimator and a logit link, for the distress variable. The invariant model was identified in line with Wu and Estabrook (2016; see Appendix D.1). We decided to use factor scores rather than sum scores to avoid tau-equivalence and to reduce measurement error (for a rationale see Appendix C.5 Part A). We pooled over the resulting factor scores and fit indices and

extracted the resulting pooled factor scores for further analyses (see Appendix D.1 for details). In sum, we could conduct the main analyses on 1130 participants, as those could be included in the imputation analyses (n = 1188) and had data for CA (n = 1139). For completeness, I provide results based on non-imputed data in online Supplement III. Next, we set out to conduct two batches of analyses.

Firstly, we aimed to shed light on the question of which RFs function (on their own) as direct effect, moderator and/or mediator for the relationship between CA and subsequent distress. To this end we started examining whether all age-14 RFs were significantly negatively associated with age-17 distress in (a) the whole sample, (b) the CA+ group, as well as (c) the CA- group. A negative association at least in the CA+ group should be a prerequisite regardless of the effect being a moderation or mediation effect. We then conducted "single" mediator and moderator models, each with one RF. For the moderation models we computed linear regression analyses with the respective age-14 RF, CA and the RF-CA interaction term (i.e. the RF multiplied with CA) as independent variables, and age-17 distress as dependent variable, to assess whether the RF moderates the relationship between CA and age-17 distress (Holmbeck, 1997). As most RFs and distress were derived from factor analyses the variables were centered at zero. To facilitate the interpretation of the binary aggression and expressive suppression RFs, we also centered those variables at zero. For the mediation models we first confirmed that the direct effect from CA on age-17 distress was significant. We then estimated a path model with CA predicting the age-14 RF (a-path) as well as age-17 distress (c'-path), and with the age-14 RF predicting age-17 distress (b-path; hence the c'-path is corrected for the effect of the RF and the b-path is corrected for the effect of CA). We additionally tested the significance of the indirect effect, which was based on the multiplication of the a-path and the b-path (MacKinnon et al., 2007). If both the indirect effect and the c'-path are significant the RF functions as partial mediator. If the indirect effect is significant, but the c'-path is not, then the RF is a full mediator (provided that the single direct effect had been significant). The mediation models were conducted in three ways, to evaluate and ensure robustness: (1) with a maximum likelihood (ML) estimator and 1000 bootstraps for the standard errors (MacKinnon, Lockwood, & Williams, 2004), (2) with a robust maximum likelihood estimator (MLR; to compute robust standard errors as described by Huber-White and scaled test statistics along the lines of Yuan-Bentler; Rosseel, 2012) and (3) with an ML estimator and simulated Monte Carlo confidence intervals (MacKinnon et al., 2004). Moreover, we applied the false discovery rate for the single RF models, to correct for multiple testing effects.

Secondly, we again aimed to investigate the question which RFs function as direct effect, moderator and/or mediator for the relationship between CA and subsequent distress, while this time taking the effects of the remaining RFs into account (i.e. "multiple" regression, mediator and moderator models). To this end we started examining whether all age-14 RFs were significantly negatively associated with age-17 distress in (a) the whole sample, (b) the CA+ group, as well as (c) the CA- group, while correcting for all other RFs. For the moderation analyses we entered all age-14 RFs, CA and all RF-CA interaction terms (i.e. 10 interactions in total) as independent variables, and age-17 distress as dependent variable to the regression model, to assess which RFs moderate the relationship between CA and age-17 distress. For the multiple mediation analysis we estimated a path model in which CA predicted the age-14 RFs (a-paths) as well as age-17 distress (c'-path), and in which all age-

14 RFs predicted age-17 distress (b-paths; hence the c'-path is corrected for the effects of the RFs and the b-paths are corrected for the effect of CA). The RFs were allowed to correlate with each other. We tested the significance of each indirect RF effect based on the multiplication of the respective a- and b-path (a*b effect). Moreover, we calculated a cumulative indirect effect adding up single indirect RF effects. Importantly, for the cumulative indirect RF effect we only included RFs that had a negative relationship with both CA and age-17 distress, when taking all other RFs into account, as only those can function as significant RF mediators (see Chapter 2 for details). The mediation models were again conducted (1) with an ML estimator and 1000 bootstraps for the standard errors (MacKinnon et al., 2004), (2) with an MLR estimator (Rosseel, 2012), and (3) with an ML estimator and simulated Monte Carlo confidence intervals (MacKinnon et al., 2004).

Importantly, all analyses were performed and will be reported with correction for gender, as gender has been shown to have a critical effect on distress (St Clair et al., 2017). The results without correction for gender are reported in Appendix E.1. Most analyses were performed in R 3.5.1 (R Core Team, 2018) and all used packages and version numbers can be found in Appendix E.2. The invariant categorical factor analysis for distress was performed in Mplus 8.2 (Muthén & Muthén, 2017). Details regarding power considerations can be found in Appendix E.3.

**Data availability.** Data for this specific paper has been uploaded to the Cambridge Data Repository https://doi.org/10.17863/CAM.46642 and is password protected. Our participants did not give informed consent for their measures to be made publicly available, and it is possible that they could be identified from this data set. Access to the data supporting the analyses presented in this paper will be made available to researchers with a reasonable request to openNSPN@medschl.cam.ac.uk.

**Code availability.** Analysis code is available on https://osf.io/nhu2z/.

## 6.3 RESULTS

### 6.3.1 Sample

Of the 1238 ROOTS participants 1139 had complete data for CA prior to age 14, and 1188 could be included in the imputation analyses for the age-14 RFs and general distress at age 14 and 17 (as they had less than 85% missing data), resulting in total in 1130 adolescents for whom we could compute the analyses (CA+ n=631, CA- n=499; female = 620, male = 510). Both gender and age did not differ between the CA+ and the CA- group; but CA+ adolescents had more often a prior psychiatric history and a lower socio-economic status (SES; see Table 6.1). Age and SES did not differ between males and females, but male adolescents less often had a prior psychiatric history.

Table 6.1
*Sample description, split for CA and gender*

| | **CA+ (n = 631)** | **CA- (n = 499)** | **$\chi^2$ /z / t (df)** | **p-value** |
|---|---|---|---|---|
| gender | Female = 358<br>Male = 273 | Female = 262<br>Male = 237 | 1.85 (1) | .17 |
| age 14* | 14.49 (0.28) | 14.48 (0.28) | -0.43 (1049.3) | .67 |
| age 17* | 17.49 (0.34) | 17.48 (0.32) | -0.48 (1015.8) | .63 |
| SES | Hard pressed = 73 | Hard pressed = 30 | 5.24 | **<.001*** |

| | | | | |
|---|---|---|---|---|
| | Moderate means = 36<br>Comfortably off = 168<br>Urban prosperity = 37<br>Wealthy achievers = 317 | Moderate means = 11<br>Comfortably off = 104<br>Urban prosperity = 41<br>Wealthy achievers = 313 | | |
| prior psychiatric history at age 14 | Yes = 199<br>No = 432 | Yes = 74<br>No = 425 | 41.54 (1) | **<.001*** |
| prior psychiatric history at age 17* | Yes = 267<br>No = 297 | Yes = 122<br>No = 345 | 48.05 (1) | **<.001*** |
| | **Female (n = 620)** | **Male (n = 510)** | **$\chi^2$ /z / t (df)** | **p-value** |
| age 14* | 14.49 (0.27) | 14.48 (0.29) | 0.61 (1027) | .54 |
| age 17* | 17.50 (0.32) | 17.47 (0.34) | 1.38 (954.71) | .17 |
| SES | Hard pressed = 51<br>Moderate means = 23<br>Comfortably off = 154<br>Urban prosperity = 34<br>Wealthy achievers = 358 | Hard pressed = 52<br>Moderate means = 24<br>Comfortably off = 118<br>Urban prosperity = 44<br>Wealthy achievers = 272 | 1.33 | .18 |
| prior psychiatric history at age 14 | Yes = 176<br>No = 444 | Yes = 97<br>No = 413 | 12.90 (1) | **<.001*** |
| prior psychiatric history at age 17* | Yes = 249<br>No = 326 | Yes = 140<br>No = 316 | 16.66 (1) | **<.001*** |

*Note.* The Pearson's $\chi^2$ tests were used for binary data and performed with Yate's continuity correction. The z-test was used for the SES variable and was conducted as asymptotic linear-by-linear association test, to account for the ordering in the data. The t-tests were used for continuous data and were conducted as Welsh's two-sample t-tests. SES was calculated based on the ACORN classification system (http://www.caci.co.uk; Morgan & Chinn, 1983). Prior psychiatric history was measured with the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Present and Lifetime Version; Kaufman et al., 1997) and included learning disabilities, clinical sub-threshold diagnoses and deliberate self-harm at age 14; and clinical sub-threshold diagnoses and deliberate self-harm, but not learning disabilities, at age 17. *Please note, the descriptive statistics are not based on the imputed data, which is why some participants have missing data on some descriptive variables, and accordingly some numbers do not add up.

### 6.3.2 Direct, Moderation and Mediation Effects for *Single* RF Models

**Direct effects.** Eight of the 10 RFs were significantly negatively associated with age-17 distress, in the CA+ group (Table 6.2 middle panel), the CA- group (Table 6.2 right panel), as well as in the whole sample (Table 6.2 left panel). Distress tolerance revealed a significantly negative effect in the CA+ group and the whole sample, but not in the CA- group. Expressive suppression was not associated with age-17 distress in any of three (sub)groups.

**Moderation effects.** None of the 10 RFs functioned as a moderator for the relationship between CA and age-17 distress (see Table 6.3).

**Mediation effects.** Eight of the 10 RFs functioned as significant RF mediators for the relationship between CA and age-17 distress (see Table 6.3), as for all those RFs CA was associated with a lower level of the RF and the RF in turn was associated with a lower level of age-17 distress. Reflection did not function as a mediator, as CA was not associated with a lower level of reflection. Expressive suppression did not function as a mediator as it was not associated with age-17 distress. Importantly, all mediating RFs functioned as partial mediators.

Table 6.2
*Single main effects of the RFs with correction for gender, for the whole sample, the CA+ and the CA- group; corrected for multiple testing*

| | ***Whole sample*** | | | | | ***CA+*** | | | | ***CA-*** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | b | b* | SE | p | $R^2$ | b | SE | p | $R^2$ | b | SE | p | $R^2$ |
| CA | 0.43 | - | .08 | **<0.001*** | 04 | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Frn | -0.35 | -0.33 | .05 | **<0.001*** | 06 | -0.35 | .06 | **<0.001*** | 06 | -0.29 | .07 | **<0.001*** | 06 |
| Fms | -0.31 | -0.29 | .04 | **<0.001*** | 06 | -0.28 | .06 | **<0.001*** | 04 | -0.31 | .06 | **<0.001*** | 07 |
| Fmc | -0.38 | -0.34 | .04 | **<0.001*** | 08 | -0.37 | .06 | **<0.001*** | 07 | -0.30 | .07 | **<0.001*** | 06 |
| Pst | -0.45 | -0.43 | .04 | **<0.001*** | 12 | -0.42 | .05 | **<0.001*** | 09 | -0.45 | .06 | **<0.001*** | 12 |
| Ngt | -0.58 | -0.56 | .05 | **<0.001*** | 14 | -0.49 | .06 | **<0.001*** | 10 | -0.67 | .07 | **<0.001*** | 18 |
| Brd | -0.48 | -0.47 | .04 | **<0.001*** | 11 | -0.44 | .06 | **<0.001*** | 09 | -0.50 | .06 | **<0.001*** | 13 |
| Rfl | -0.29 | -0.28 | .05 | **<0.001*** | 05 | -0.25 | .07 | **<0.001*** | 03 | -0.31 | .07 | **<0.001*** | 06 |
| Dst | -0.24 | -0.21 | .04 | **<0.001*** | 04 | -0.29 | .06 | **<0.001*** | 05 | -0.09 | .07 | 0.18 | 03 |
| Agg | -0.28 | -0.26 | .04 | **<0.001*** | 06 | -0.24 | .05 | **<0.001*** | 05 | -0.28 | .06 | **<0.001*** | 06 |
| Exp | 0.00 | 0.02 | .04 | 0.98 | 02 | 0.04 | .05 | 0.42 | 01 | -0.01 | .06 | 0.81 | 02 |
| D14 | 0.65 | 0.63 | .04 | **<0.001*** | 22 | 0.61 | .05 | **<0.001*** | 19 | 0.66 | .06 | **<0.001*** | 25 |

*Note.* b = unstandardized regression (slope) coefficient, b* = unstandardized regression (slope) coefficient corrected for CA; SE = standard error, p = p-value, $R^2$ = variance explained, represented here as percentage. Of note, SE, p and $R^2$ belong to the effects that are not corrected for CA. CA = childhood adversity, CA+ = adolescents with CA, CA- = adolescents without CA; Frn = friendship support; Fms = family support; Fmc = family cohesion; Pst = positive self-esteem; Ngt = negative self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression; D14 = age-14 distress. The belonging results that are not corrected for gender can be found in Appendix E.1.

Table 6.3

*Single moderation and mediation effects of the RFs with correction for gender; corrected for multiple testing*

| | **Moderation: interaction effect** | | | | **Mediation: indirect effect** | | | |
|---|---|---|---|---|---|---|---|---|
| | b | SE | p | %$R^2$ | b | SE | p | MC CI |
| Frn | -0.06 | 0.10 | 0.72 | 08 | 0.04 | 0.02 | **<0.05*** | .01-.08 |
| Fms | 0.03 | 0.09 | 0.76 | 08 | 0.05 | 0.02 | **<0.01*** | .02-.08 |
| Fmc | -0.07 | 0.09 | 0.72 | 09 | 0.12 | 0.02 | **<0.001*** | .08-.17 |
| Pst | 0.04 | 0.08 | 0.72 | 13 | 0.11 | 0.02 | **<0.001*** | .07-.17 |
| Ngt | 0.20 | 0.09 | 0.24 | 16 | 0.10 | 0.03 | **<0.001*** | .05-.15 |
| Brd | 0.07 | 0.09 | 0.72 | 13 | 0.06 | 0.02 | **<0.05*** | .02-.11 |
| Rfl | 0.08 | 0.09 | 0.72 | 07 | 0.01 | 0.01 | 0.41 | -.01-.04 |
| Dst | -0.17 | 0.09 | 0.24 | 06 | 0.05 | 0.01 | **<0.001*** | .02-.08 |
| Agg | 0.03 | 0.08 | 0.76 | 08 | 0.06 | 0.02 | **<0.001*** | .03-.10 |
| Exp | 0.05 | 0.08 | 0.72 | 04 | -0.00 | 0.01 | 0.59 | -.02-.01 |
| D14 | -0.06 | 0.08 | 0.72 | 23 | 0.16 | 0.03 | **<0.001*** | .09-.23 |

*Note.* b = unstandardized regression (slope) coefficient, SE = standard error, p = p-value, %$R^2$ = percentage variance explained. MC CI = Monetcarlo confidence interval. Frn = friendship support; Fms = family support; Fmc = family cohesion; Pst = positive self-esteem; Ngt = negative self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression; D14 = age-14 distress. The belonging results that are not corrected for gender can be found in Appendix E.1.

### 6.3.3 Direct, Moderation and Mediation Effects for *Multiple* RF Models

The theoretical models that demonstrate multiple moderator and mediator models are depicted in Figure 6.2.

**Direct effects.** When splitting the group in CA+ and CA-, the findings diverged notably. In the CA+ group six RFs (Table 6.4 middle panel), and in the CA- group two RFs had a significantly negative effect on age-17 distress (Table 6.4 right panel; see also Appendix E.4 for variance inflation factors). In the overall sample five RFs were significantly negatively associated with age-17 distress, namely: family cohesion, positive self-esteem, negative self-esteem, brooding and aggression (Table 6.4 left panel). Yet, of those five RFs only brooding had a significantly negative effect in both the CA+ and the CA- group. Positive self-esteem had a significantly negative effect in the CA+ and a marginally negative effect in the CA- group ($p$ = 0.07).

**Moderation effects.** CA was as expected positively associated with distress. Negative self-esteem revealed a significant moderation effect (see Figure 6.3; or Appendix E.5 for details). Yet, negative self-esteem had no relationship with age-17 distress in the CA+ group, and a significantly negative relationship with age-17 distress in the CA- group (see Table 6.4). Hence, negative self-esteem had the opposite effect as an RF. Friendship support, family cohesion and distress tolerance had a negative relationship with age-17 distress in the CA+ group, and no relationship with age-17 distress in the CA- group. Yet, their moderation effects did not reach significance (friendship support $p = 0.07$, family cohesion $p = 0.09$, and distress tolerance $p = 0.09$).

**Mediation effects.** We first computed the multiple mediation models and examined which RFs had a negative relationship with both CA (a-path) and distress (b-path; when controlling for the b-paths of all other RFs and the CA effect), as only those can function as mediating RFs. Eight RFs qualified (see Figure 6.3). Expressive suppression and reflection did not qualify as they did not have a negative relationship with age-17 distress. Of the eight potential RF mediators three functioned as significant partial mediators, namely: positive self-esteem, negative self-esteem, and aggression. Brooding revealed a marginal mediation effect ($p = 0.06$). All eight RFs together had a significant cumulative mediation effect (of note, we also included RFs with nonsignificant paths in the cumulative mediation effect as long as the paths had the correct slope sign/directionality).

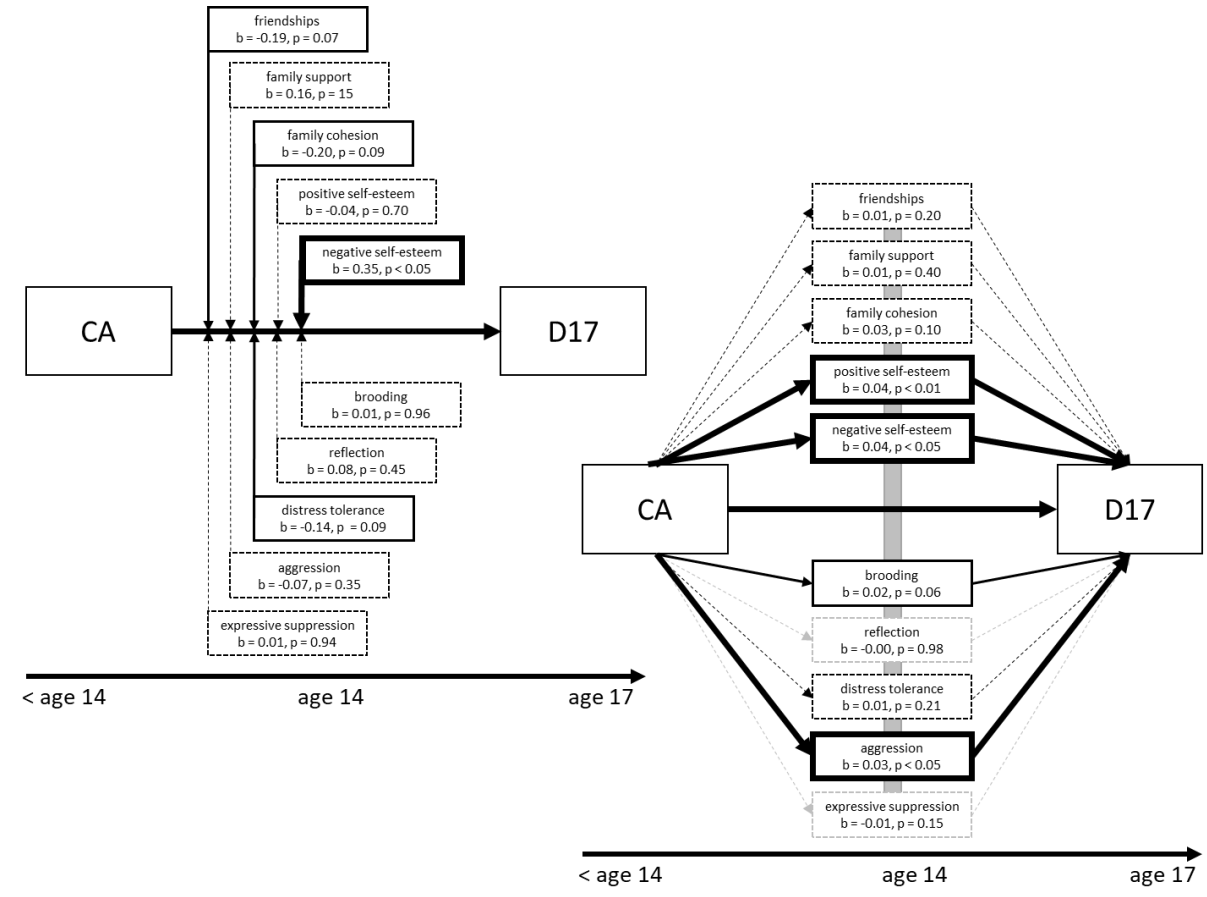



*Figure 6.3.* The left panel depicts a multiple moderator model and the right panel the multiple mediator model, both corrected for gender. CA = childhood adversity, D17 = distress at age 17. Bold, solid lines = significant effects; thin, solid lines = marginal effects ($0.05 \geq p < 0.10$); black, dashed lines = non-significant effects. Grey, dashed lines =

nonsignificant effects of those variables that are not included in the cumulative mediation effect, for reasons detailed in the main text. The grey bar in the mediation model indicates that the RFs are allowed to correlate with each other.

Table 6.4
*Multiple main effects of the RFs with correction for gender, for the whole sample, CA+ and the CA- group*

| | ***Whole sample*** | | | | ***CA+*** | | | ***CA-*** | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | b | b* | SE | p | b | SE | p | b | SE | p |
| CA | 0.25 | - | .07 | **<0.001*** | - | - | - | - | - | - |
| Frn | -0.08 | -0.07 | .05 | 0.12 | -0.15 | .07 | **<0.05*** | 0.03 | .08 | 0.66 |
| Fms | -0.04 | -0.05 | .06 | 0.50 | 0.01 | .08 | 0.86 | -0.15 | .08 | **0.06** |
| Fmc | -0.12 | -0.09 | .06 | **<0.05*** | -0.17 | .08 | **<0.05*** | 0.03 | .08 | 0.76 |
| Pst | -0.17 | -0.16 | .05 | **<0.001*** | -0.18 | .07 | **<0.01*** | -0.14 | .08 | **0.07** |
| Ngt | -0.20 | -0.20 | .07 | **<0.01*** | -0.06 | .09 | 0.54 | -0.40 | .10 | **<0.001*** |
| Brd | -0.18 | -0.18 | .06 | **<0.01*** | -0.17 | .09 | **<0.05*** | -0.18 | .09 | **<0.05*** |
| Rfl | 0.01 | 0.00 | .06 | 0.93 | 0.02 | .08 | 0.82 | -0.05 | .08 | 0.50 |
| Dst | -0.07 | -0.05 | .04 | 0.11 | -0.12 | .06 | **<0.05*** | 0.03 | .06 | 0.68 |
| Agg | -0.12 | -0.11 | .04 | **<0.01*** | -0.14 | .05 | **<0.01*** | -0.07 | .06 | 0.26 |
| Exp | 0.05 | 0.06 | .04 | 0.16 | 0.05 | .05 | 0.27 | 0.05 | .05 | 0.39 |
| gender | -0.11 | -0.11 | .08 | 0.15 | -0.08 | .11 | 0.45 | -0.15 | .11 | 0.20 |
| % $R^2$ | | | | 19% | | | 17% | | | 22% |

*Note.* b = unstandardized regression (slope) coefficient, b* = unstandardized regression (slope) coefficient corrected for CA; SE = standard error, p = p-value, % $R^2$ = percentage variance explained. Of note, SE, p and %$R^2$ belong to the effects that are not corrected for CA. CA = childhood adversity, CA+ = adolescents with CA, CA- = adolescents without CA; Frn = friendship support; Fms = family support; Fmc = family cohesion; Pst = positive self-esteem; Ngt = negative self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression. The belonging results that are not corrected for gender can be found in Appendix E.1.

## 6.4 DISCUSSION

We aimed to shed light on the effects that best describe *how* RFs reduce mental health problems after CA. To this end we looked at direct, moderation and mediation effects. We first investigated models separately for the ten RFs, so called *single* RF models. We found that eight out of 10 RFs had a significantly negative direct effect on age-17 distress in both the CA+ and the CA- group, and thus also in the overall sample. While no RF revealed a significant moderation effect, as many as eight of the ten RFs revealed a significant mediation effect. Next, we tested models including all ten RFs, so called *multiple* RF models, to safeguard overestimating *single* RF effects and to take the ecological validity sufficiently into account. This time, five RFs had a significantly negative direct effect in the overall sample. Yet, of those five, only brooding revealed a significant effect in both groups. Positive self-esteem had a significant effect in the CA+ group, but only a marginal effect in the CA- group. The moderation effects of friendship support, family cohesion and distress tolerance did not reach significance, despite that all three RFs had a significantly negative relationship with age-17 distress in the CA+ group, and no effect in the CA- group. Positive self-esteem, negative self-esteem and aggression functioned as significant mediators. Brooding revealed a borderline mediation effect. All in all, our findings provide predominant support for RFs to function as mediators rather than as

moderators, regardless of investigating *single* or *multiple* RF models. Below, I shall first embed the moderation and then the mediation results within the wider literature.

Previous literature has demonstrated that distress tolerance (Banducci et al., 2017) and family climate (similar to family cohesion; Klasen et al., 2015) moderate the relationship between CA and subsequent distress, in single RF models (see also Table 6.5). In other words, *intra*-personal distress acceptance and *inter*-personal cohesion with the next of kin were found to reduce subsequent distress particularly in those adolescents with a history of CA. We did not find corresponding effects in *single* RF models. Moreover, in our *multiple* RF model, both the moderation effect of distress tolerance and of family cohesion only approached significance. Whether such marginal effects should or should not be interpreted, has been debated by scientists since decades (Greenland et al., 2016). Neyman and Pearson (1928), two key developers of statistical hypothesis testing, stated that "it is doubtful whether the knowledge that *P* was really .03 (or .06) rather than .05 . . . would in fact ever modify our judgment" (p. 201 in Neyman & Pearson, 1928) and explain that "the tests should only be regarded as tools which must be used with discretion and understanding, and not as instruments which in themselves give the final verdict" (p. 232 in Neyman & Pearson, 1928). To err on the side of caution, I will here *not* interpret the marginal moderation effects and will thus not draw conclusions regarding differences between adolescents with and without CA. However, I do interpret the fact that distress tolerance and family cohesion reduced subsequent distress significantly in the group of adolescents with CA, even after correcting for the other RFs. In sum, given our fairly inconclusive moderation findings, our results diverge slightly from the reviewed findings, which indicated preliminary support for the moderation effects of distress tolerance and family cohesion. Yet, our findings do to some degree reveal a similar conclusion. That is, when taking an ecological stance – taking into account the existence of *multiple* potential RFs – distress tolerance and family cohesion seemed to be important factors for decreasing subsequent distress in adolescents with prior exposure to adversity. Whether the protective value of distress tolerance and family cohesion differs eventually significantly between groups of adolescents with and without CA (or not) needs to be re-tested in future research.

For social support the literature is not conclusive, including some studies that do find (Klasen et al., 2015), and others that do not find support for a moderation effect (Shahar & Henrich, 2015). We found no moderation effect in the *single* RF model, and only a marginal effect in the *multiple* RF model. Yet, as for distress tolerance and family cohesion, friendship support reduced subsequent distress only significantly in the group of adolescents with CA, and not in the group without CA. As before, while I interpret the significant promotive effect of friendship support in the CA+ group, I refrain from drawing conclusions regarding the marginal difference between the two groups (i.e. the marginal moderation effect). Based on the results of the reviewed studies, one may wonder whether friendship support reduces internalizing symptoms particularly after the exposure to family-related CA (e.g. parental psychopathology as in Klasen et al., 2015), but less so for CA that has taken place outside the family (e.g. rocket attacks as in Shahar & Henrich, 2015; or bullying as in van Harmelen et al., 2016). Hence, it may be the case that friendships are particularly beneficial in maintaining or increasing mental health after exposure to family-related CA. This would make sense, as a disadvantageous family environment may in itself be less supportive and sometimes even hazardous, in which case adolescents may benefit

from seeking support outside of the family. Such a conjecture would align with cross-domain support matching (Gore & Aseltine Jr., 1995). Yet, some research suggests that cross-domain support matching has not proven to be effective (Gore & Aseltine Jr., 1995).

While the literature did reveal some support for RF moderation effects, our research revealed at best very weak, marginal interaction effects, rendering our moderation results inconclusive. One possible account may come from our sample size, which was smaller than for example the sample used in prior literature for family cohesion and social support ($N$ > 1500). Another account may come from the fact that our CA variable was binary, and therefore potentially less sensitive than an index reflecting CA severity or frequency. The binary CA variable may have, in addition to the restricted sample size, limited the explanatory power. A third explanation may, as above explained for friendship support, be that RF moderation effects depend on the specific type of CA. It seems unlikely that our mental health outcome variable has contributed to the divergence from previous findings, as the reported previous literature either used anxiety or depression symptoms as outcome variable, which are both captured in our transdiagnostic distress index.

Table 6.5
*Moderation and mediation effects of single and multiple RF models, found in the literature*

| | **Moderation Effect** | **Mediation Effect** |
|---|---|---|
| Frn | ✓(Klasen et al., 2015)<br>×(Shahar & Henrich, 2015)[*1] | ×(van Harmelen et al., 2016) |
| Fms | ✓(Shahar & Henrich, 2015)[*1]<br>×(Shahar & Henrich, 2015) [*1]<br>×(van Harmelen et al., 2016) | ✓(van Harmelen et al., 2016) |
| Fmc | ✓(Klasen et al., 2015) | ✓(Finan et al., 2015)[*2]<br>×(Finan et al., 2015)[*2] |
| Pst | ✓(Dubow et al., 2012)[*3] | |
| Ngt | ✓(Dubow et al., 2012)[*3] | |
| Brd | ×(Boyes et al., 2015)[*4] | ✓(Boyes et al., 2015)[*4]<br>×(Gaté et al., 2013) |
| Rfl | ×(Boyes et al., 2015)[*4] | ✓(Boyes et al., 2015)[*4]<br>×(Gaté et al., 2013) |
| Dst | ✓(Banducci et al., 2017) | |
| Agg | | ✓(You & Lim, 2015)<br>×(You & Lim, 2015) |
| Exp | ×(Boyes et al., 2015)[*4] | ✓(Boyes et al., 2015)[*4] |

*Note.* Frn = friendship support; Fms = family support; Fmc = family cohesion; Pst = positive self-esteem; Ngt = negative self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression. ✓ = this effect was tested and found; × = this effect was tested but not found; *effects found in multiple RF models, taking into account the following RFs: [1] = friend support, family support, school support; [2] = family cohesion, communication with the mother, communication with the father; [3] = self-esteem, academic grades, positive parenting; [4] = rumination, expressive suppression, cognitive reappraisal (although the respective moderation effect was only corrected for the main effects, not the interaction effects of the other RFs). Importantly, a detailed synthesis and narrative for all the findings that are contained in Table 6.5, can be found in Chapter 2, the original systematic review from which this information is taken.

With regard to mediation effects, previous literature has demonstrated that aggression (You & Lim, 2015) and brooding (Boyes et al., 2015) function as intermediate variables between CA and subsequent mental health problems. Yet, the literature is not fully conclusive and contains some findings that do not support the suggested mediation effects (see e.g. Gaté et al., 2013, for brooding; and You & Lim, 2015, for aggression). While the literature revealed a mediation effect for aggression in a *single*

RF model, our research found support in *single* as well as *multiple* RF models. Moreover, the literature revealed a mediation effect for brooding in a *multiple* RF model, whereas we found predominant support for brooding in the *single* RF model and only marginal support in the *multiple* RF model. Interestingly, while self-esteem has previously been shown to function as a moderator (Dubow et al., 2012), our results rather indicate that positive and negative self-esteem are important mediators for the relationship between CA and age-17 distress. Furthermore, we found that the cumulative RF mediation effect was particularly robust and strong. Overall, the probably most notable finding of our study is that while all revealed moderation effects were weak and marginal, most mediation effects were strong and robust. This could for example be related to power. As explained above, our binary CA variable had a limited explanatory power, which may have predominantly disadvantaged the highly power sensitive moderation effects (see our power calculations in Appendix E.3). However, the fact that we mainly found support for mediation than for moderation could also simply be the correct result, given the statistical nature of the two different effects. That is, RF moderators need to have a stronger effect on distress in the CA+ than in the CA- group, whereas RF mediators can have a comparably strong effect on distress in the CA+ and the CA- groups.

Besides the discussion on the relevance of CA-RF matching, a highly interesting consideration is how transdiagnostic RF effects are. For many of the most robust RFs there seems to be some preliminary transdiagnostic evidence. For instance, for ruminative brooding there is a broad and solid literature that supports its relationship with depression related disorders, anxiety related disorders and obsessive compulsive disorder, as well as some evidence for its association with bulimia and alcohol abuse (Wahl et al., 2019). There is preliminary but good support for self-esteem, particularly for its effects on internalizing related disorders (Keane & Loades, 2017). Moreover, anger (i.e. a form of aggression) was found in conjunction with other emotion regulation skills to function as transdiagnostic effect on internalizing and externalizing symptoms (McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). Similarly, social support (Cohen, Danielson, Adams, & Ruggiero, 2016), family functioning (Santesteban-Echarri et al., 2018), and distress tolerance (Cohen et al., 2016) have been suggested to function as transdiagnostic effects on mental distress symptoms. Yet, knowledge on anger or aggression, social support, family functioning and distress tolerance seems so far limited, or seems at least to lack synopsis, and more meta-synthesis is needed. Unsurprisingly, self-esteem, brooding and emotion-regulation have already been found to be successful intervention targets (Kovacs et al., 2006; Millar & Donnelly, 2013; Watkins, 2015), particularly for interventions aimed at reducing internalizing disorders and/or increasing mental well-being.

Our findings suggest that improving levels of many of the tested RFs in early adolescence may improve mental health during later adolescence and young adulthood. However, in which way does this knowledge inform translational research or (upon replication) clinical settings? Technically both moderation and mediation effects may lend themselves well for prevention and treatment. Speculatively, one could assume that mediation effects may be particularly advantageous for prevention, as they require a negative impact of the CA exposure on the RF as well as an increased risk of mental distress after CA. Hence, if prevention would start to enhance the RF early on, the deleterious effect of CA on the RF could be disrupted, and the enhanced RF could in turn foster or

increase mental health at an early stage, ideally before the onset of mental health problems. For moderation effects, such a consideration is less relevant as CA does not need to directly relate to the RF. Important is however, that the RF moderator reduces subsequent distress more in adolescents with than without exposure to CA. Such an effect is therefore more easily detected when the RF has no effect on distress in the group of adolescents without CA. Yet, RFs that are not also protective for adolescents without CA should not be targeted in interventions aimed at both individuals with and without CA. Moreover, such RFs should ideally also not be targeted when a CA history is unknown and cannot be assessed, as it is sometimes the case in school or community settings. When the aim is to prioritise interventions aimed at all adolescents regardless of a history of CA, one should focus on those RFs that are successful in reducing distress significantly in both the group of adolescents with and without CA. Based on our findings one would need to enhance low brooding and potentially positive self-esteem. Importantly, our findings only suggest potentially relevant factors for interventions, but we first need evidence of how effective those RFs actually are, when being targeted in interventions, before we can be certain about their effects.

A related and still largely unanswered question is whether it is more efficient to target and enhance RFs that adolescents have already acquired, or to target RFs that adolescents have not yet acquired well. Theoretically both seems plausible. RF skills or resources that are already present may be particularly efficient targets, as they may be relatively easy to be detected and capitalized on, but may induce limited change. Therefore, one may wonder whether those RFs may be particularly helpful for acute but limited improvement in mental health. RF skills and resources that are only immaturely developed may be particularly effective targets, as they may be difficult and time-consuming to acquire, but may induce more change once acquired. Therefore, one may wonder whether those RFs may be particularly helpful for slow but substantial improvement in mental health. According to Ellis, Bianchi, Griskevicius, and Frankenhuis (2017) interventions that focus on disadvantaged skills have not yet proven to be helpful and have particularly established how hard it is to induce effective skill-set changes following CA. Another important consideration when choosing RF targets, also discussed by Ellis and colleagues (2017) is that it may be dangerous to “de-claw the cat” (p. 566 in Ellis et al., 2017). In other words, it may be safer to improve already existing skills, rather than trying to induce new or revert skills, particularly when those are likely to disadvantage the adolescents in some contexts. For instance, while in general it may be advantageous to improve distress tolerance skills in adolescents (i.e. reduce distress sensitivity), it may for some adolescents be disadvantageous or even dangerous. Taking adolescents in violent home environments as an example, it seems logical that low distress tolerance, as in being sensitive and alert when situations generate friction, may actually be advantageous. Therefore, it seems highly important to (a) be aware of the underlying type of the protective RF effect, so that appropriate expectations can be set, and to (b) be aware for which particular group (or adolescent) the RF target at hand is suitable. Both requirements for choosing RF targets for translational research clearly need further scientific evaluation.

Our research is of course not without limitations. Besides sampling restrictions, such as an above-average prosperity (Goodyer et al., 2010), the study also had limitations regarding the measurement. For example, some RFs were based on subscales of the same questionnaire (which

may result in higher correlations due to equivalent answer options), and not all questionnaires were developed for the specific RFs (e.g. expressive suppression). For future research it would be advisable to use scales particularly set out for the specific RFs, as this is likely to increase the psychometric quality. Along those lines, the dichotomous CA variable which we have used may be restricted in range, which may, as explained above, have limited the explanatory power. Future research may besides CA exposure also want to assess the severity and frequency of the exposure (Schlechter et al., 2019). Moreover, in addition to a retrospective assessment, a prospective assessment would be ideal. Yet, an advantage of our CA variable is that CA was assessed by caregiver report, whereas most RFs (eight out of 10) and distress were assessed via adolescent self-report. Therefore, the adversity variable cannot be directly biased by the adolescents' mood state. Another potential limitation may come from the missing data treatment. Here we used predictive mean matching (for ordered categorical items) and logistic algorithms (for binary items) to estimate missing values. We imputed the RFs and distress on the item level and then ran factor analyses on the imputed items, as the lower the level on which the imputation is performed, the more information is available and thus bias is less likely. As we did not perform the imputations on the latent (factor score) level, we could not include interactions between CA and the RFs in the imputation analysis. Yet, research has shown that moderation analyses based on imputed data are generally more precise when interaction terms of the variables are included in the imputation model (White, Royston, & Wood, 2011). To provide the reader with as much information as possible, we also computed all analyses based on non-imputed data. However, given the item-level imputation approach, which we have chosen to prioritize imputation precision, we cannot disentangle whether differences in moderation results between imputed and non-imputed data are the result of a potentially insufficiently complex imputation model or because of selection bias in the non-imputed sample. Furthermore, it is important to remind the reader that all results are solely derived from between-person analyses, indicating group level effects, and are thus likely to not directly translate to the individual (idiographic) level. However, the probably most important limitation is that we did not have data on mental distress and RFs prior to CA and we therefore do not know whether the increased mental distress and decreased RF levels in adolescents with CA (as compared to adolescents without CA) were perhaps already (to some degree) present prior to CA. For future research it would be ideal, if those effects could be assessed and scrutinized.

In sum, we showed that improving levels of many of the tested RFs in early adolescence may improve mental health during later adolescence and young adulthood. Specifically, enhancing self-esteem, low brooding, and low aggression may disrupt the deleterious relationship between CA and mental distress, and thereby improve or stabilize mental health. Our findings further suggest that for settings in which we cannot assess adversity, one may want to focus on enhancing low brooding, as low brooding seems to reduce subsequent mental health problems regardless of a history of CA. Translational research may be advised to match the purpose of the studied intervention closely with the specific type of the RF effect of interest.

In the next chapter, I aim to investigate those two RFs that seemed most important in this and the previous chapters: high self-esteem and low brooding. More specifically, I shall study those two RFs before, during and after a stress-inducing exam period in medical students. This way, we can find out

whether the two RFs change from before to after the natural stressor, co-evolve with changes in mental distress, and mitigate mental distress during and/ or after the stress-inducing exams.

# CHAPTER 7

# On the Dynamics and Mutual Relations of Mental Distress, Brooding and Self-Esteem from Before to After Naturally Occurring Exam Stress

About one in five young people experience mental distress in the form of anxiety and depression (Randall, Corp, Self, & Office for National Statistics, 2016; World Health Organization, 2018). “Early identification of such problems – and, when necessary, early intervention or timely management – is critically important. . . . In the absence of appropriate support and intervention, such problems may continue, worsen or lead to mental illness.” (report of the World Health Organization, 2018, page 1). Resilience factors (RFs), such as high self-esteem and low brooding, mitigate mental distress in the face of stressful experiences (see Chapter 2 or Fritz, de Graaff, et al., 2018). The literature contains a considerable amount of knowledge on RFs that mitigate concurrent and subsequent mental distress (Afifi & MacMillan, 2011; Braithwaite et al., 2017; Marriott et al., 2014; Traub & Boynton-Jarrett, 2017; Wright et al., 2013). Yet, studies investigating RFs over the time span from before to after a stressor, i.e. stress that causes or triggers mental distress, are concerningly scarce (Danese, 2019). However, knowledge on mental distress and RFs before and during the stressor is crucial, as it (a) is otherwise impossible to determine whether mental distress and RFs are affected by the stressor (Danese, 2019; Kalisch et al., 2015), and (b) is necessary to identify those RFs that are potentially promising prevention targets. Equally, knowledge on mental distress and RFs during and after the stressor is essential, as this (a) enables us to identify whether mental distress and RFs recover after the stressor (Masten, 2011), and (b) indicates which RFs may be promising treatment targets at times of stress.

Recent research suggests that medical students are at-risk for the development of mental distress. A meta-analysis based on 122,356 medical students from 43 countries has shown that the prevalence rate for depressive symptoms was 27.2 percent (range individual studies: 1.4 - 73.5; Rotenstein et al., 2016). This prevalence rate was higher than for population representative peers of a similar age (e.g. compared to the 9.3 percent prevalence, found for young adults aged 18 to 25, in the US National Survey on Drug Use and Health; N = 67,500; Rotenstein et al., 2016). Besides depression, anxiety and general distress levels have also been found to be elevated in students performing medical degrees (when compared to population representative samples; Dyrbye, Thomas, & Shanafelt, 2006). Unsurprisingly, exam stress has been identified as a potential trigger for mental ill-health in medical students (Dyrbye, Thomas, & Shanafelt, 2005; Dyrbye et al., 2006; Yusoff et al., 2013). Building on this knowledge, I designed the RESIST study in which we assessed perceived stress, mental distress and RFs in Cambridge University medical students before, during and after their yearly exam period (Fritz, Stochl, Kievit, van Harmelen, & Wilkinson, 2020). We found, as expected, that perceived stress and mental distress were lower before than during exams, but higher before than after exams (see for a summary Appendix F.1, or for details Fritz, Stochl, Kievit, et al., 2020). Hence, on the group level, the exams functioned indeed as a stressor. Here, we aim to take our research endeavor a step further, through exploring whether RFs co-evolve with and mitigate mental distress over the time span from

before to after the stress-inducing exams. More specifically, we shall focus on the two RFs low brooding and high self-esteem, due to their well-known promotive effects on mental health in the general population (see Chapter 5 or Fritz, Stochl, Goodyer, et al., 2020; Millar & Donnelly, 2013; Watkins, 2015), and in individuals exposed to stress (see Chapter 2; Boyes et al., 2015; Dubow et al., 2012; Chapter 3 or Fritz, Fried, et al., 2018; Chapter 4 or Fritz et al., 2019).

Brooding is generally defined as "'moody pondering', negative self-evaluative thinking (e.g., 'Why can't I handle things better?') and comparative thinking about the self (e.g. 'Why do I have problems other people don't have?')" (p. 2 in Watkins & Roberts, 2020), in a passive, abstract and rather uncontrollable manner (McEvoy et al., 2018; Treynor et al., 2003; Watkins & Roberts, 2020). Research has shown that a high level of brooding is associated with several negative mental health consequences, including a higher risk for depression related disorders, post-traumatic stress disorder, anxiety disorders, suicidal behaviour and non-suicidal self-injury, eating disorders, insomnia, substance abuse, and psychosis (Watkins & Roberts, 2020). In contrast, low levels of brooding have been shown to promote good mental health and in particular to mitigate internalizing symptoms (Watkins, 2015). In other words, while a high level of brooding functions as a risk factor and can pose detrimental consequences for mental health, a low level of brooding equally functions as RF and can prevent or reduce mental distress.

Self-esteem is sometimes described as a positive self-concept. Greenberg (2008) explains that questions such as "Do you care about being a . . . good romantic partner, a good parent, and a good friend? Do you desire to make valuable contributions to your profession? Do you want to behave morally and competently in the domains of your life?" reflect concerns and desires that stem from self-esteem (p. 48 in Greenberg, 2008). A high level of self-esteem has been shown to be related to less mental distress, depression, anxiety, and eating pathology, as well as to more well-being (Bos, Muris, Mulkens, & Schaalma, 2006; Greenberg, 2008). While self-esteem is commonly defined as an RF (for a review see Shean, 2015; or Traub & Boynton-Jarrett, 2017), a low level of self-esteem or self-worth is part of the DSM 5 criteria for depression ("Feelings of worthlessness"; American Psychiatric Association, 2013). Hence, whereas low self-esteem is assumed to contribute to low mood, a high level of self-esteem may protect against low mood levels.

Both brooding and self-esteem have already been found to be successful intervention targets (Millar & Donnelly, 2013; Watkins, 2015), particularly for interventions aimed at reducing internalizing disorders and/or increasing mental well-being. Accordingly, we will approach low brooding and high self-esteem from a strength-based mental health perspective and understand them as RFs. In sum, we know that both brooding and self-esteem can be considered to have transdiagnostic effects, and can successfully be targeted in mental health interventions. However, we know little about how brooding and self-esteem evolve from before to after a stressful experience and whether they co-evolve with mental distress. Therefore, we here take on this challenge by investigating three research questions:

First, we investigate whether brooding and self-esteem levels change from before to during and after the exams. Such knowledge will shed light on the stress responsivity (or stability) of brooding and self-esteem. Second, we investigate whether brooding and self-esteem co-evolve with mental distress. This will provide an indication for whether brooding and self-esteem predominantly predict change in

mental distress, whether mental distress predominantly predicts change in brooding and self-esteem, or whether changes in the two resilience factors and mental distress are dependent on each other (also called mutualistic change). Third, we investigate whether brooding and self-esteem mitigate increase in mental distress during exams, as well as support recovery from mental distress after exams. As introduced earlier, RFs before a stressor that mitigate increase in distress during the stressor and/ or support recovery of mental distress after the stressor may be valuable prevention targets; RFs during the stressor that support recovery of mental distress after the stressor may be valuable treatment targets (at times of stress). In sum, answers to our research questions may help inform about the nature of brooding and self-esteem and about the time at which it may be most fruitful to leverage them. Such knowledge may then aid translational research and eventually help to inform student support services, mental health services as well as resilience and transdiagnostic mental health theory.

### 7.1.1 Hypotheses

For the first question, whether brooding and self-esteem change from before to after the exams, we set out to test the five most plausible change trajectories (see also Figure 2 in Fritz, Kievit, et al., 2020): negative homeostasis (RFs change in the opposite direction as mental distress, i.e. RF levels are relatively moderate before exams, low during exams and high after exams); positive homeostasis (RFs change in the same direction as mental distress, i.e. RF levels are relatively moderate before exams, high during exams and low after exams); growth (RFs improve over time); deterioration (RFs deteriorate over time); and the no-change trajectory (RFs stay stable over time).

For the second question, whether brooding and self-esteem change mutualistically with mental distress, we expect that the two RFs influence change in mental distress negatively. However, we have no expectation as to whether mental distress influences change in brooding and self-esteem, as we do not know whether the two RFs decrease, stay stable, or increase. Therefore, we have no specific expectation as to whether we will find a mutualistic change effect.

The third question, whether brooding and self-esteem mitigate increase in mental distress during as well as support recovery of mental distress after the exams, is mainly exploratory. Yet, as for the second question, we assume that brooding and self-esteem influence the change in mental distress negatively: i.e. students with greater RF levels before exams will, on average, increase less in mental distress during exams as well as decrease more in mental distress after exams; and students with greater RF levels during exams will decrease more in distress after exams. All aims, hypotheses and proposed analyses are further detailed in our preregistration (see Fritz, Kievit, et al., 2020): https://osf.io/wxv8q/.

## 7.2 METHODS

### 7.2.1 Design

RESIST is a cohort study, with three measurement occasions and a within-subject (natural) stress manipulation (i.e. the exam period). Occasion 1 took place in a non-exam period, during the University

term (February and March 2018). Occasion 2 took place during the end-of-year exam period (approximately April to June 2018, depending on the timing of the exam period). Occasion 3 took place after the exam period, at the end of the term for year 6 students (for whom exams are earlier; approximately end of May to Mid-July 2018), and in the summer vacation/autumn term for year 1 to 5 students (approximately Mid-August to Mid-October 2018). At all three occasions students were asked to complete a survey, containing a series of online questionnaires. At occasion 2, students were provided with the questionnaires three weeks before their first final exam. The questionnaires had to be completed before taking the last final exam. This way all participants were exposed to the same type of naturally-occurring external stressor.

### 7.2.2 Sample and Procedure

We recruited first to sixth year Cambridge medical students over the age of 18. The students received the online link to the questionnaire (survey software: REDCap) via email. To prevent double partaking, we sent personalized emails (i.e. unique links) to the students. Participants received monetary reimbursement for partaking (online vouchers: £5 for occasion 1, £7 for occasion 2, £5 for occasion 3). Participants who completed all three occasions were additionally entered in a prize draw (prize: 5x£50 online vouchers). RESIST was approved by the Cambridge Psychology Research Ethics Committee (PRE.2017.096). Further details regarding the procedure are reported in Fritz, Stochl, Kievit, and colleagues (2020).

### 7.2.3 Demographic and Clinical Characteristics

We assessed eight demographic and clinical variables: Academic course, year of academic education, gender, age, ethnicity, parental educational level, psychotherapeutic treatment, and psychopharmacology intake (i.e. prescribed drugs).

### 7.2.4 Mental Distress

We assessed mental distress with the 12 item version of the General Health Questionnaire (GHQ-12; Hankins, 2008). The GHQ-12 provides a broad indication of mental health across the spectrum from low to high mental distress, but does not act as a measure of diagnosable mental illness. The self-report items assess topics such as concentration, sleep, or happiness (measured on a 4-point Likert scale). The GHQ was previously found to have a Cronbach's alpha between .78 and .95 (Jackson, 2007). The GHQ-12 had previously a mean area under the ROC curves of .88 (Goldberg et al., 1997). In our sample, the GHQ-12, in the remainder referred to as mental distress, had a good reliability (Cronbach's alpha: o1 = 0.88, o2 = 0.89, o3 = 0.90; coefficient omega: o1 = 0.89, o2 = 0.89, o3 = 0.91). Higher sum scores indicate a higher level of mental distress.

### 7.2.5 Resilience Factors

**Brooding.** We assessed ruminative brooding with the 5 item brooding subscale of the Ruminative Responses Scale (RRS; 22 items in total; Treynor et al., 2003). The self-report items assess brooding levels for questions such as why things do not work out better or why other people do not have

comparable problems (previously reported brooding subscale Cronbach's alpha = .77; Treynor et al., 2003). In our sample, brooding had an acceptable reliability (Cronbach's alpha: occasion (o) 1 = 0.75, o2 = 0.79, o3 = 0.77; coefficient omega: o1 = 0.76, o2 = 0.80, o3 = 0.78). Higher sum scores indicate a lower level of brooding.

**Self-esteem.** We assessed self-esteem with 10 items of the standardized Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965). The self-report items assess positive self-esteem levels such as being capable of doing things well, and negative self-esteem levels such as feeling useless (previously reported Cronbach's alpha = 0.88; Gray-Little, Williams, & Hancock, 1997; Rosenberg, 1965). In our sample, self-esteem had an excellent reliability (Cronbach's alpha: o1 = 0.93, o2 = 0.94, o3 = 0.92; coefficient omega: o1 = 0.93, o2 = 0.94, o3 = 0.92). Higher sum scores indicate a higher level of self-esteem.

### 7.2.6 Analysis

For the first question, exploring which trajectory describes the change in brooding and self-esteem best, we conducted a series of latent growth models (LGMs). We conducted each of the two LGMs, that is one for brooding and one for self-esteem, four times:

- M1 → once as freely estimated trajectory (modelling the actual change; i.e. the unconstrained model) with two anchoring slope loadings,
- M2 → once as negative homeostasis trajectory (slope loading = s: $s_{o1}>s_{o2}<s_{o3}$, $s_{o1}<s_{o3}$) or as positive homeostasis trajectory ($s_{o1}<s_{o2}>s_{o3}$, $s_{o1}>s_{o3}$), depending on which of the two can be estimated given the two anchoring slope loadings,
- M3 → once as growth trajectory ($s_{o1}<s_{o2}<s_{o3}$) or as deteriorating trajectory ($s_{o1}>s_{o2}>s_{o3}$), again depending on which of the two can be estimated given the two anchoring slope loadings, and
- M4 → once as no-change trajectory (latent mean slope value fixed to 0) with the same two anchoring slope loadings (see also Figure 2 in Fritz, Kievit, et al., 2020, for the depiction of the potential trajectories).

We then compare the LGMs using different absolute fit indices (i.e. the confirmatory fit index [CFI]: ≥ .95 = good; the Tucker-Lewis fit index [TLI]: ≥ .95 = good; the standardized root mean square residual [SRMR]: ≤ .08 good; and the root mean square error of approximation [RMSEA]: < .08 = acceptable, < .06 = good), as well as several comparative fit indices (i.e. the Akaike information criterion [AIC]: lower = better; the Bayesian information criterion [BIC]: lower = better; and AIC and BIC model comparison weights [$AIC_w$ and $BIC_w$]: the higher the weight the better the model fit as compared to the other tested models). Additionally, we used $Chi^2$ tests to compare the best fitting model against the no-change trajectory model, to find out whether mean change can be considered significant.

For the second question, whether brooding and self-esteem change mutualistically with mental distress, we conducted a series of bivariate latent change score models (BLCSMs; as described in Kievit et al., 2018). We conducted each of the two BLCSMs, that is one for brooding and one for self-esteem, three times: once for change from before to during exam, once for change from before to after exams, and once for change from during to after exams. This allowed for direct comparisons without

estimating overly complex models. We used the standard BLCSM estimation (for details see our preregistration Fritz, Kievit, et al., 2020; or Kievit et al., 2018).

The third research question, whether brooding and self-esteem mitigate increase in mental distress during as well as support recovery of mental distress after the exams, was evaluated based on the directionality (i.e. positive or negative slope sign) and significance ($p < 0.05$) of the mutualistic coupling effects of the previously described BLCSMs.

Fourth, we estimated a set of exploratory regression models to find out whether brooding and self-esteem predict absolute mental distress levels (as opposed to change in mental distress). We deemed this additional, exploratory (i.e. not preregistered) analysis to be important, as it enables us to understand whether RFs predominantly mitigate change in mental distress (in the face of a stressor), or predominantly explain the absolute "severity" of mental distress. Equally, such knowledge indicates whether mental distress predominantly predicts change in self-esteem and brooding (in the face of a stressor), or predominantly explains the absolute mean level of self-esteem and brooding. To be able to explore the prediction of absolute mean levels we had to omit autoregressive paths, as the correction of auto-regressive paths would per definition have resulted in the prediction of change scores.

We conducted all analyses with a Full Information Maximum-Likelihood (FIML) estimator, to account for missing data. The specific sample size varied per analysis, as it depends on the particular combination of occasions and the investigated variable(s). As robustness analyses, we re-estimated the analyses (a) with additional missingness predictors as auxiliary variables, to facilitate the FIML estimation (n = 451; see Appendix F.2), as well as (b) on the full sample, excluding all students who have missingness for at least one of the three analyses variables (i.e. brooding, self-esteem and mental distress) on at least one of the three occasions (n = 218; see Appendix F.3). All analyses were performed in R version 3.5.1 (R Core Team, 2017). The used R packages are reported in Appendix F.4.

**Data availability.** The anonymized data used for the analyses of this chapter has been uploaded to the Cambridge Data Repository, at https://doi.org/10.17863/CAM.57030.

**Code availability.** Analysis code is available on https://osf.io/8s27p/.

## 7.3 RESULTS

### 7.3.1 Sample

Students were approximately uniformly distributed over all six academic years (that is, 56 – 93 students per year; see Table 7.1). Fifty-seven percent of the students were female (1 percent preferred not to answer) and 58 percent were white. Most students were between 18 and 23 years old and had parents with a higher education after secondary school. About 14 percent of the students received psychotherapeutic treatment and 11 percent received psychopharmaceutic treatment in the six months prior to occasion 1. Of note, none of the analysis variable combinations posed a multicollinearity problem (the highest variance inflation factor was 1.8; see Appendix F.5).

Table 7.1
*Demographic and clinical statistics for the overall sample*

| **Academic year** | | |
|---|---|---|
| 1= 21% (93), 2= 18% (83), 3= 15% (66), 4= 20% (88), 5= 12% (56), 6= 14% (65) | | |
| **Age*** | | |
| 18-20 = 38% (170), 21-23 = 44% (196), 24-26 = 14% (65), 27-29 = 03% (13), 30+ = 01% (4) | | |
| **Gender**** | **Education Mother[1]** | **Education Father**,[1]** |
| female = 57% (259) | yes = 80% (359) | yes = 82% (369) |
| male = 41% (185) | no = 19% (88) | no = 17% (77) |
| prefer not to say = 1% (6) | unknown = 1% (4) | unknown = 1% (4) |
| **Ethnicity***** | **Psychological Treatment[2]** | **Psychiatric Drug Treatment[2]** |
| white = 58% (263) | yes = 14% (61) | yes = 11% (49) |
| non-white = 41% (184) | no = 86% (390) | no = 89% (402) |

*Note.* Exact sample sizes are depicted between brackets. *Three students did not answer this question. **One student did not answer this question. ***Four students did not answer this question. Due to the missingness, some percentages do not add up. [1]Further/ higher education after secondary school. [2]Treatment for the 6 months prior to occasion 1.

### 7.3.2 Research Question 1

Which trajectory describes change in brooding and self-esteem best?

**Brooding.** For brooding, the no-change trajectory described the data best (Model 4 in Table 7.2). Yet, the fit did not differ significantly from the next best trajectory, the negative homeostasis trajectory ($Chi^2$ difference = 0.72, df = 1, $p$ = 0.40). Despite the very small change in brooding (negative homeostasis), we conclude that brooding is best described by a no-change trajectory (see Figure 7.1).

**Self-esteem.** For self-esteem, the negative homeostasis trajectory described the data best (Model 2 in Table 7.2). This suggests that self-esteem is higher before than during the exam period, but higher after the exams than beforehand (self-esteem slope loadings/ levels: before estimated at 0.8, during scaled to 0, after scaled to 1; Model 2 in Table 7.3). As the negative homeostasis trajectory differed significantly from the no-change trajectory ($Chi^2$ difference = 12.79, df = 1, $p$ < 0.001), self-esteem seemed to change significantly over the three occasions.

Table 7.2
*Latent growth model fit*

| Model | AIC | BIC | CFI | TLI | RMSEA | SRMR | $Chi^2$ (df) | BICw | AICw |
|---|---|---|---|---|---|---|---|---|---|
| **Brooding** | | | | | | | | | |
| M1* | 4909.47 | 4934.14 | 1.00 | 1.00 | 0.00 | 0.01 | 1.788 (3) | 07.4% | 32.3% |
| M2* | 4909.47 | 4934.14 | 1.00 | 1.00 | 0.00 | 0.01 | 1.788 (3) | - | |
| M3* | 4910.62 | 4935.29 | 1.00 | 1.00 | 0.00 | 0.02 | 2.936 (3) | 04.1% | 18.2% |
| M4* | 4908.62 | 4929.17 | 1.00 | 1.00 | 0.00 | 0.02 | 2.929 (4) | 88.5% | 49.5% |
| M1* vs M4* | | $Chi^2$ difference = 0.72, df = 1, $p$ = 0.40 | | | | | | | |
| **Self-esteem** | | | | | | | | | |
| M1* | 7076.36 | 7100.98 | 0.98 | 0.98 | 0.11 | 0.04 | 18.44 (3) | 99.8% | 99.9% |
| M2* | 7076.36 | 7100.98 | 0.98 | 0.98 | 0.11 | 0.04 | 18.44 (3) | - | - |
| M3* | 7090.93 | 7115.54 | 0.96 | 0.96 | 0.15 | 0.04 | 33.00 (3) | 00.1% | 00.1% |
| M4* | 7093.42 | 7113.94 | 0.95 | 0.96 | 0.14 | 0.04 | 37.50 (4) | 00.1% | 00.0% |
| M1* vs M4* | | $Chi^2$ difference = 12.79, df = 1, $p$ < 0.001 | | | | | | | |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. AIC = Akaike information criterion, BIC = Bayesian information criterion, CFI = confirmatory fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, df = degrees of freedom, p = p-value. *the

variance for the latent slope fixed to 0, to render it non-negative. In case of the no-change trajectory having the best fit, we compared it against the model with the next best fit.

Table 7.3
*Latent growth model summary*

| Model | Slope ld. o1 | Slope ld. o2 | Slope ld. o3 | Intercept mean | Slope mean | Residual var. o1 | Residual var. o2 | Residual var. o3 | Intercept slope cv. |
|---|---|---|---|---|---|---|---|---|---|
| **Brooding** | | | | | | | | | |
| M1* | 0.97 | 0.00 | 1.00 | 14.07 | 0.17 | 3.69 | 3.69 | 3.69 | -0.27 |
| M2* | 0.97 | 0.00 | 1.00 | 14.07 | 0.17 | 3.69 | 3.69 | 3.69 | -0.27 |
| M3* | 0.00 | 0.00 | 1.00 | 14.17 | 0.10 | 3.69 | 3.69 | 3.69 | 0.05 |
| M4* | 2.01 | 0.00 | 1.00 | 14.20 | 0.00 | 3.70 | 3.70 | 3.70 | -0.17 |
| **Self-esteem** | | | | | | | | | |
| M1* | 0.81 | 0.00 | 1.00 | 45.70 | 1.93 | 24.27 | 24.27 | 24.27 | -10.17 |
| M2* | 0.81 | 0.00 | 1.00 | 45.70 | 1.93 | 24.27 | 24.27 | 24.27 | -10.17 |
| M3* | 0.00 | 0.00 | 1.00 | 46.81 | 0.80 | 25.04 | 25.04 | 25.04 | -10.53 |
| M4* | 0.18 | 0.00 | 1.00 | 47.19 | 0.00 | 25.26 | 25.26 | 25.26 | -11.57 |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. ld. = loading, o = occasion, var. = variance, cv. = covariance. *the variance for the latent slope fixed to 0, to render it non-negative.

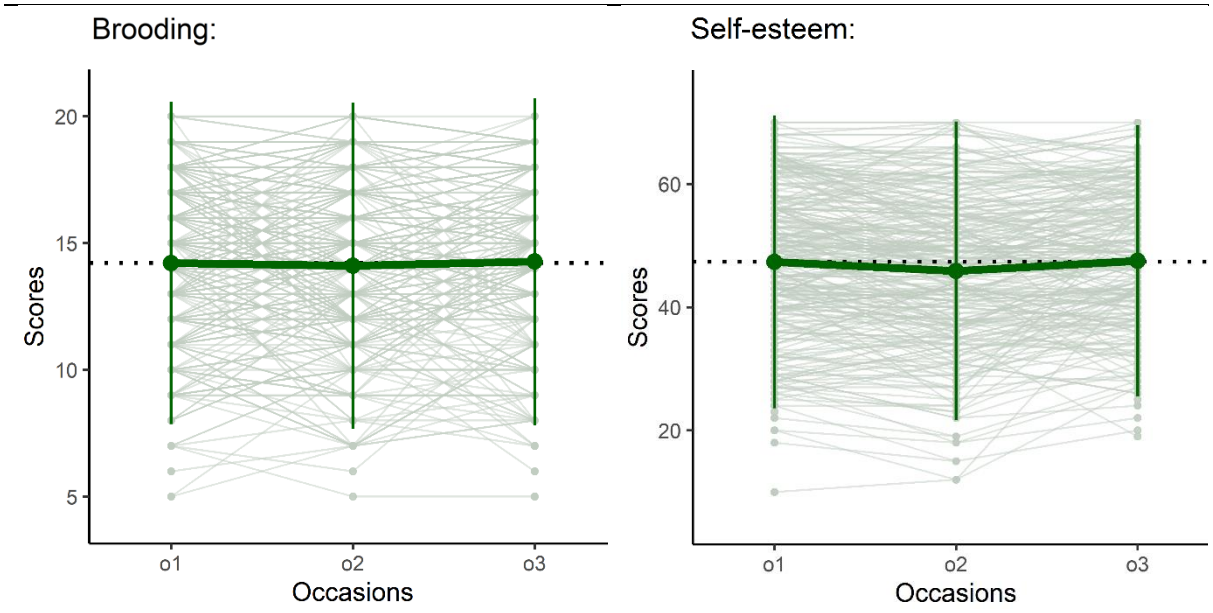



*Figure 7.1.* RF change trajectories for brooding (left) and self-esteem (right). The faded grey lines indicate person-level trajectories. The green line indicates the group-level sum score trajectory. The dotted black line represents the group-level sum score for the respective RF at occasion 1, solely to enhance the comparison with the other occasions. For exact mean levels and standard deviations see Appendix F.6.

### 7.3.3 Research Question 2

Do brooding and self-esteem change mutualistically with mental distress?

**Brooding.** For before to during exams, brooding was negatively associated with the rate of change in mental distress, and mental distress was negatively associated with the rate of change in brooding. That is, lower brooding was on average associated with a lower increase in mental distress,

and higher mental distress was on average associated with more decrease in low brooding (i.e. with more increase in abstract, negative-focussed and repetitive thinking; Figure 7.2 upper left panel). For before to after, as well as during to after, exams, brooding was not associated with the rate of change in mental distress, and mental distress was not associated with the rate of change in brooding (Figure 7.2 middle and lower left panel). In sum, a significant mutualistic effect between brooding and mental distress was only found from before to during exams, not for the other time frames (see Appendix F.6 for exact coefficients).

**Self-esteem.** For before to during exams, self-esteem was negatively associated with the rate of change in mental distress (Figure 7.2 upper right panel). That is, higher self-esteem before exams was on average associated with a lower increase in mental distress during exams. For before to after exams, self-esteem was not associated with the rate of change in mental distress (Figure 7.2 middle right panel). For during to after exams, self-esteem was negatively associated with the rate of change in mental distress (Figure 7.2 lower right panel). That is, higher self-esteem during exams was on average associated with more decrease in mental distress after exams. Mental distress was not associated with the rate of change in self-esteem for any of the time-point combinations. In sum, we did not detect mutualistic effects between self-esteem and mental distress, rather, self-esteem seemed to be more predictive for change in mental distress than vice versa (see Appendix F.6 for the exact coefficients).

### 7.3.4 Research Question 3

Do brooding and self-esteem mitigate increase in mental distress during as well as support recovery of mental distress after the exams?

**Brooding.** Low brooding before exams mitigated increase in mental distress during exams, but neither brooding before nor during exams fostered decreases of mental distress after exams. Hence, in our naturalistic study, the effects of low brooding seemed predominantly preventative.

**Self-esteem.** Self-esteem before exams mitigated increase in mental distress during exams, but did not foster decreases of mental distress after exams. Self-esteem during exams did foster decreases of mental distress after exams. Hence, self-esteem seemed to have a preventative effect before the exams, as well as a promotive effect during exams which fostered recovery of mental distress after the exams.

### 7.3.5 Exploratory Research Question 4

Do brooding and self-esteem predict (absolute) mental distress severity during as well as after the exams?

**Brooding.** For brooding the regression model results, predicting absolute mean levels, were comparable to the above described BLCSM results, predicting change in mean levels. The only notable difference is that while mental distress before and during exams was not associated with the rate of change in brooding after exam stress, mental distress before and during exams was associated with brooding mean levels after exams (see Figure 7.3 left panel). In other words, higher mental distress before and during exams was on average associated with more abstract, negative-focussed and

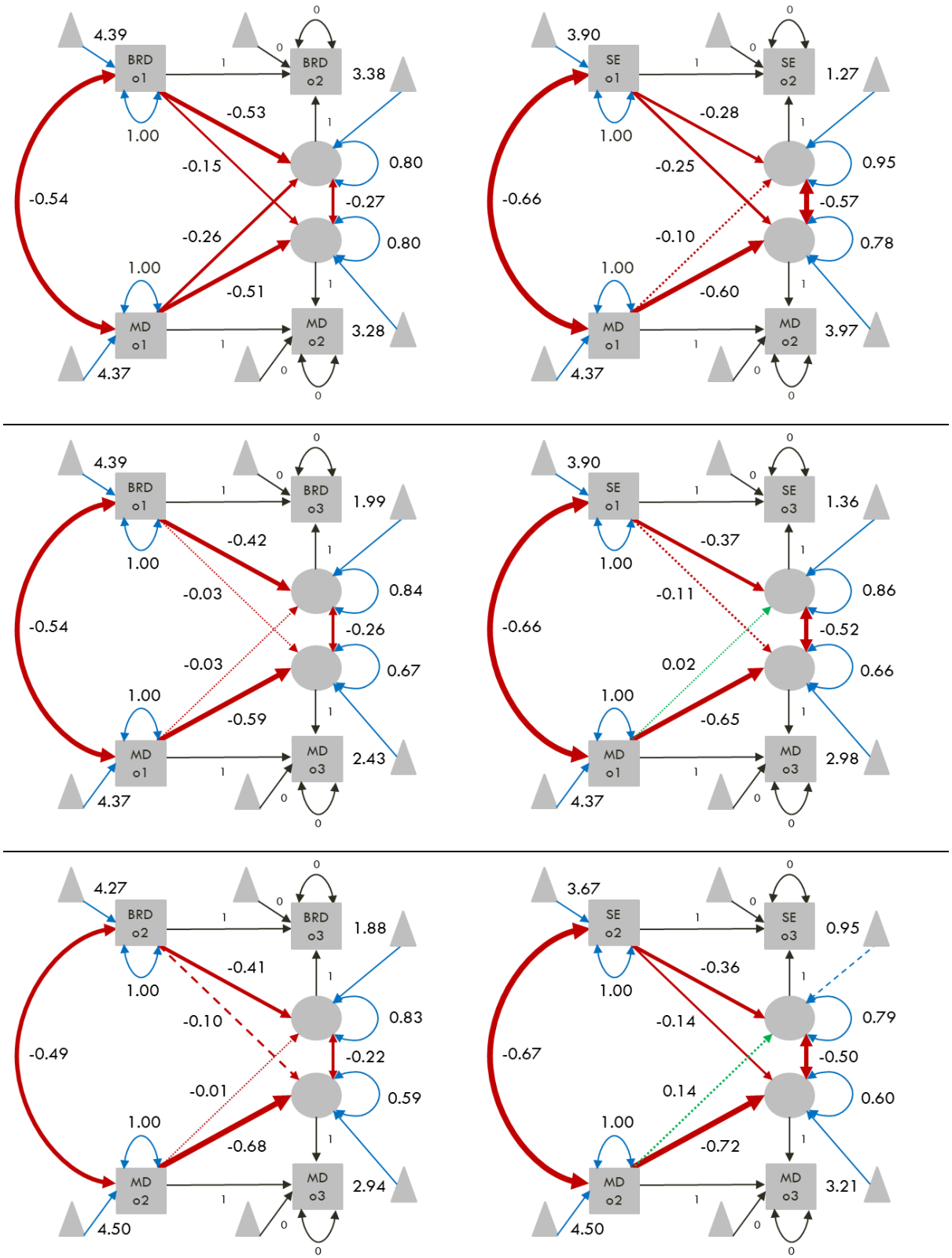



*Figure 7.2.* Bivariate latent change score models. Left panel = brooding, right panel = self-esteem, upper panel = occasion 1 to 2, middle panel = occasion 1 to 3, lower panel = occasion 2 to 3. MD = mental distress, BRD = brooding, SE = self-esteem, o = occasion. Double-headed arrows = (co)variances, one-headed arrows = intercepts and (auto)regressions. Red arrows = negative relationships, green arrows = positive relationships, black arrows = fixed parameters, blue arrows = intercepts and variances. The thickness of the green and red arrows indicates the relationship strength. Solid line = significant association ($p < .05$), dashed line = marginal association ($.05 \geq p < .10$), dotted line = non-significant association ($p \geq .10$). Grey squares = manifest variables, grey circles = latent variables, grey triangles = intercepts. Exact estimates can be found in Appendix F.6.

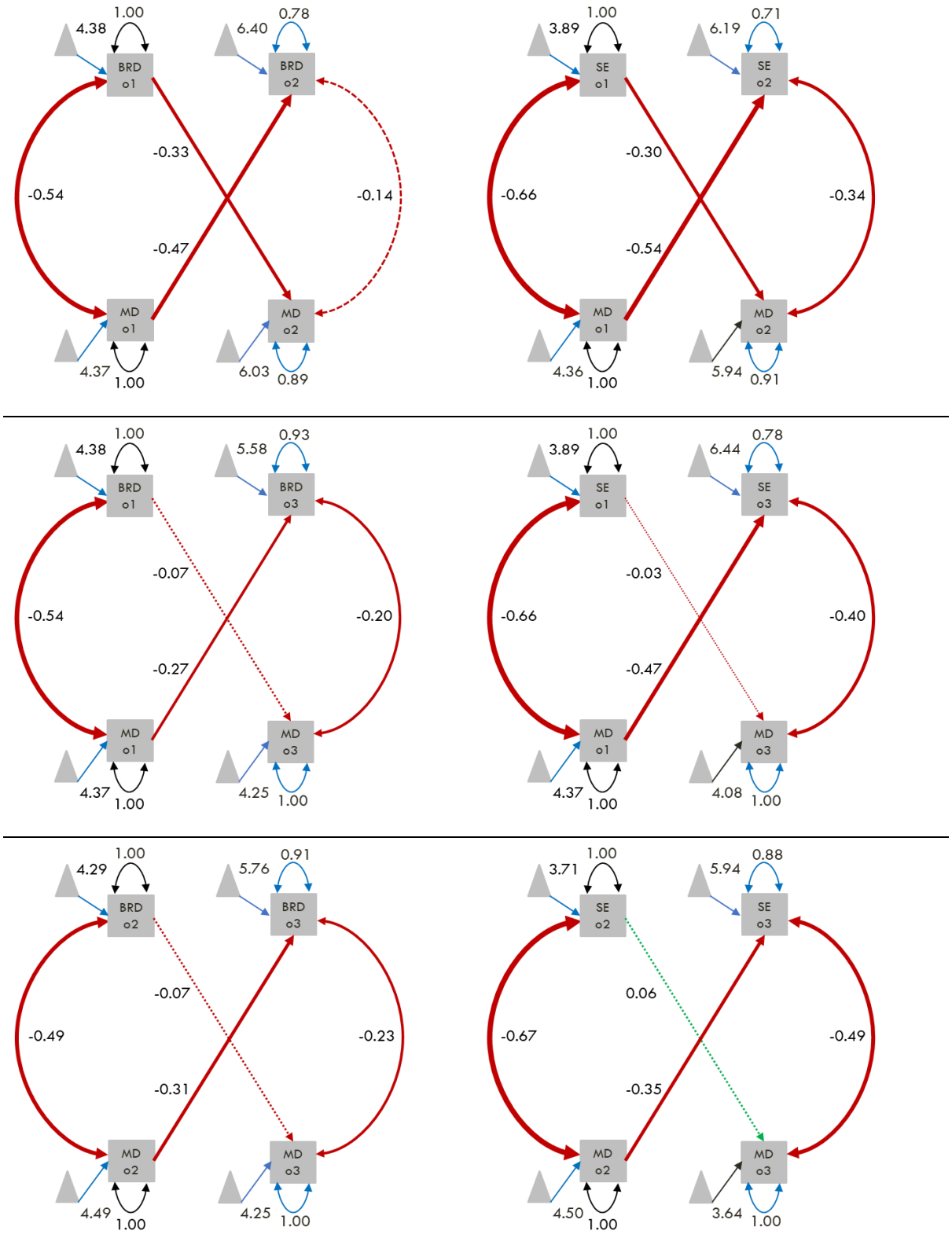



*Figure 7.3.* Regression models. Left panel = brooding, right panel = self-esteem, upper panel = occasion 1 to 2, middle panel = occasion 1 to 3, lower panel = occasion 2 to 3. MD = mental distress, BRD = brooding, SE = self-esteem, o = occasion. Double-headed arrows = (co)variances, one-headed arrows = intercepts and regressions. Red arrows = negative relationships, green arrows = positive relationships, black arrows = fixed parameters, blue arrows = intercepts and variances. The thickness of the green and red arrows indicates the relationship strength. Solid line = significant association ($p < .05$), dashed line = marginal association ($.05 \geq p < .10$), dotted line = non-significant association ($p \geq .10$). Grey squares = manifest variables, grey triangles = intercepts. Of note, we did not include autoregressive paths, to predict absolute mean levels rather than change scores.

repetitive thinking during and after exams.

**Self-esteem.** For self-esteem the results differed notably from the BLCSM results. While in the change models self-esteem predominantly predicted change in mental distress (rather than the other way around), in the regression models only self-esteem before exams was associated with absolute mental distress levels during exams (i.e. higher self-esteem was on average associated with less mental distress). Self-esteem was not associated with absolute distress levels at the other two time-point combinations (see Figure 7.3 right panel). In contrast, mental distress predicted absolute self-esteem levels for all time-point combinations (i.e. higher mental distress was on average associated with less self-esteem). Taken together this suggests that self-esteem predominantly predicts change in but not absolute levels of mental distress, while mental distress predicts absolute levels of but not change in self-esteem.

## 7.4 DISCUSSION

We aimed to find out whether brooding and self-esteem (a) change in response to stress-inducing exams, (b) change mutualistically with mental distress, and (c) mitigate increase in mental distress during as well as foster recovery of mental distress after the exams. In sum, we found that (a) self-esteem, but not brooding, changed from before to after the exams, (b) brooding had a mutualistic coupling effect with mental distress from before to during exams (not for the other occasions), while self-esteem had a predominant effect on change in mental distress (rather than vice versa), and that (c) both brooding and self-esteem mitigated increase in mental distress during exams, but only self-esteem fostered recovery of mental distress after exams. I shall now discuss the interpretation of the results for both RFs and embed them within the wider literature.

Low brooding before exams mitigated increase in mental distress during exams, while high mental distress before exams facilitated increase in negative, repetitive brooding during exams, which suggests mutualistic interactions during this time period. Additionally, brooding before exams explained the absolute mental distress severity during exams. Brooding did neither explain change in nor absolute mental distress after exams. This suggests that brooding is likely to be most fruitful as prevention target when increases in mental distress can be expected. The crucial question is, however, whether it is possible to intervene on and reduce brooding, to increase its resilience enhancing effect. Hoorelbeke and colleagues (2015) found that in undergraduates, cognitive control training (CCT) could successfully be implemented to reduce brooding in the face of exams (Hoorelbeke, Koster, Vanderhasselt, Callewaert, & Demeyer, 2015). Moreover, Cook, Mostazir and Watkins (2019) found preliminary evidence for the preventative effect of brooding on students' mental health in a translational intervention study. More specifically, they showed that internet-based rumination-focused cognitive behavioural therapy (RFCBT; compared against treatment as usual) was successful in reducing depression risk in undergraduates with elevated levels of brooding (Cook et al., 2019). Hence, preventative interventions such as CCT and RFCBT seem to be successful in reducing high brooding or fostering low brooding, which in turn reduces the risk of subsequent distress symptoms. Taken together, we conclude that

brooding (or perhaps rumination generally) may potentially be a fruitful prevention target to aid resilient responses to exam stress and/ or natural stressors in general, and should be further examined in translational research.

Self-esteem was best described by a negative homeostasis change trajectory and thus had the opposite change trajectory as mental distress. In other words, self-esteem seemed to have the highest level in times when mental distress was (on average) lowest. While, self-esteem mitigated increases in mental distress during exams, as well as fostered recovery of mental distress after exams, mental distress did not predict change in self-esteem. In contrast, when predicting absolute mean levels instead of change in mean levels, mental distress predominantly predicted self-esteem rather than the other way around. Speculatively this may mean that self-esteem does not so much affect the overall mental distress severity but affects how much mental distress changes in response to a stressor, whereas mental distress may affect the general self-esteem level but does not seem to affect how self-esteem changes in response to a stressor. In sum, given that self-esteem both mitigated increase in distress during exams and fostered recovery of mental distress after exams, translational research should test whether self-esteem is indeed a viable target for prevention as well as for treatment at times of high stress. Unsurprisingly, self-esteem has already been found to be a successful treatment target for promoting mental health (Millar & Donnelly, 2013). Yet, to the best of our knowledge less evidence exists for self-esteem as prevention target (Bos et al., 2006). Moreover, there is preliminary evidence that suggests that high self-esteem is only helpful if it is stable (Bos et al., 2006; Greenberg, 2008). Hence, translational intervention research is clearly needed to show whether self-esteem is most fruitful as prevention target to aid resilient responses to (natural) stressors, as treatment target (at times of stress), or as both; and mechanistic intervention research is needed to confirm whether self-esteem is indeed only helpful if it is generally stable and does not fluctuate from moment to moment.

When placing our findings within the already existing scientific evidence, some higher-level research questions arise both with regard to resilience and transdiagnostic mental health theory, as well as with regard to medical student mental health. Regarding resilience and transdiagnostic mental health theory an interesting research aim for future studies may be to re-conduct our study, this time specifically assessing diagnoses or trait-level distress, instead of recent, short-term distress. On the short-term, for quick stress relief, RFs that mitigate negative emotional states may already be helpful, but on the long run, RFs that mitigate negative affective traits may be particularly advantageous. Moreover, given that we have specifically looked at a mental health continuum, our research falls short on identifying those students who would qualify for a diagnosable mental illness. On the one hand, our approach is advantageous as it (a) solely relies on mental distress severity and therefore includes below symptom-threshold cases, and (b) naturally accommodates comorbidity, which is in line with transdiagnostic mental health theory. On the other hand, however, our distress continuum approach may make us forget that some level of anxiety, depression and distress symptoms may actually be advantageous (Nesse, 2004). Thus, future research should attempt to find out whether (intervening on) self-esteem and brooding also foster(s) well-being, happiness and educational success. In other words, I believe that future research is needed to shed light on the question whether increasing or fostering RFs would indeed result in people being overall better off.

Regarding medical student mental health one may wonder whether looking to improve psychological RFs to relieve students from mental distress may be sufficient, or whether such an approach may miss out on the potential root cause of the experienced distress. Recent research not only suggests that medical students have higher mental distress than age-matched peers (Dyrbye et al., 2006; Rotenstein et al., 2016), but also that mental health problems rise during medical education. For example, a recent report on clinical medicine students of the University of Cambridge (i.e. students from the same student body as in our study) found that most medical students with mental health problems, namely 61.8 percent, started encountering those problems after starting with medical school (Jacob et al., 2020). This is particularly interesting as according to reports from the World Health Organization fifty percent of all mental health problems start manifesting earlier, namely before or during adolescence (World Health Organization, 2018). Thus, as discussed in the previous paragraphs, promoting student mental health may well be a realistic endeavour (e.g. via RFCBT); yet, one may wonder whether some qualitative research into the potentially unfavourable impact of university education in general or medical education in particular may be necessary too.

Besides theoretical limitations, our research has several practical limitations. For example, only 31 percent of the medical student body took part. Moreover, our study had a notable decline in participation rate (during compared to before exams = 39%; after compared to before exams = 37%). Yet, we tried to reduce the potential attrition bias through using a FIML estimator throughout our analyses. As our students were solely from the University of Cambridge, generalization is likely to be limited both with regard to other population groups (e.g. due to an on average higher socio-economic status and a higher level of education), and with regard to student populations studying a different subject, or the same subject at a different university. Moreover, there are several proximal factors that may impact RFs and mental distress, which we could not control for (e.g. homesickness, wrong judgement of the own potential, negative relationships with supervisors, burden of shaping a professional identity; Crook, 2020; Jacob et al., 2020; for students from abroad: language barriers, new nutrition, culture, and climate; Crook, 2020).

Additionally, we have some analytic limitations to report. First, we conducted our analyses on sum-score rather than item level, to enhance power. This may go on the cost of measurement precision and implies metric invariance between measurement occasions, as in a sum-score coefficient all items have the same contribution (i.e. loading) to the coefficient across measurement occasions. Yet, we conducted additional robustness analyses (see Appendix F.7) which indicate that metric invariance could not be rejected for the mental distress, brooding and self-esteem variables. Second, we did not conduct BLCSMs based on all three occasions. Technically, this may increase Type I errors. However, this was necessary to avoid identification issues (due to the higher complexity of BLCSMs with three time-points) and to enhance power. Third, we encountered estimation problems when additionally including auxiliary variables (i.e. variables explaining missingness) in the models. This is likely a result of the limited sample size in relation to the rather complex models, as we could conduct the models with a FIML estimator without auxiliary variables. Hence, our models account to some degree for missingness, but do not rely on additional model-unrelated auxiliary variables. For completeness, I report the results based on models with auxiliary variables as well as based on models only including

the complete sample (i.e. including those participants who had data for the analyses variables at all three occasions; *N* = 218) in Appendices F.2 and F.3. Moreover, for completeness we also split self-esteem into high positive and low negative self-esteem, as was done in previous reports (see Chapters 3 to 6), which mainly revealed the same findings (see details in Appendix F.8).

In sum, we found that both high self-esteem and low brooding mitigated increases in mental distress during exams in medical students, suggesting that both have a potentially promising prevention effect. Additionally, we found that self-esteem during exams fostered recovery of mental distress after exams, suggesting that self-esteem may also be a fruitful target for treatments at times of stress. Based on our findings I recommend future research to (a) find out whether our findings replicate in independent student samples as well as in other at-risk populations (e.g. fire brigade, police force or soldiers), (b) repeat our research while specifically disentangling whether RFs have different effects on disorder diagnoses or mental distress traits than on short-term distress states, (c) repeat our research with well-being, happiness, professional success and life satisfaction as outcome measures, (d) test brooding and self-esteem in translational research as *prevention* targets and (e) explore particularly self-esteem as a target for treatments at times of stress.

In the next and last Chapter, I shall summarize the main findings of this doctoral thesis, relate them to each other, and discuss the overall limitations and implications in depth.

# CHAPTER 8

# Reflections on the Complex Picture of Psychosocial Factors That Promote Mental Health in Young People

Allen Frances, the former director of the working group that tried to establish diagnostic guidelines for psychological illnesses (the DSM-IV – Diagnostic and statistical manual; American Psychiatric Association, 2000), stated that "We are at the epicycle stage of psychiatry where astronomy was before Copernicus and biology before Darwin." (see p. 23-24 in Nesse, 2019). Obviously, knowledge on mental health problems needs to proliferate and advance, and my doctoral research is at the best a humble contribution. Yet, I believe that the only way to enhance understanding is to keep going with robust and replicable research. Therefore, I will here offer some points of reflection and implications of my doctoral research, will shed light on the most important limitations, and will discuss directions and ideas for follow-up research. First however, I shall start with a summary for each chapter.

## 8.1 SYNOPSIS OF MY DOCTORAL RESEARCH

The first contribution of my thesis, i.e. Chapter 2, is a preregistered systematic review which had the aim of identifying empirically-supported resilience factors (RFs) that reduce the risk of mental health problems in young people subsequent to childhood adversity (CA). In the review, I focused on amenable RFs that function on social, emotional, cognitive and behavioural levels, and can thus be targeted in preventative or therapeutic psychosocial interventions. The literature search identified 1969 studies, of which 22 were included. Overall, I found empirical support for 13 of 25 individual-level (e.g., high self-esteem, low rumination), six of 12 family-level (e.g., high family cohesion, high parental involvement), and one of five community-level (i.e., high social support) RFs. Those findings cautiously suggest that (preventative) interventions that improve the levels of these RFs may reduce the probability of mental health problems following CA. The findings also indicate that most of the studies investigated RFs in isolation from each other and only the vast minority of the studies tested the effectiveness of multiple RFs at the same time. Moreover, none of the studies had specifically tested the interactions between RFs, although some studies acknowledged that RFs seem to interrelate strongly.

Building on the notion that examining individual RFs may not be sufficient to understand the system that protects individuals from developing mental health problems, in Chapter 3, I studied the interrelations of those 10 RFs that were identified in Chapter 2 and were assessed in our population-based adolescent cohort (N = 1139; age 14). More specifically, I used network analysis to establish a complex system of three inter-personal (high friendship support, high family support, and high family cohesion/climate) and seven intra-personal RFs (high positive self-esteem, high distress tolerance, low negative self-esteem, low reflective rumination, low ruminative brooding, low aggression, and low expressive suppression). I then examined whether those RF networks differ between groups of adolescents with (n = 638) and without a history of CA (n = 501), expecting that the RFs will be related

to each other in both group networks, but that the two group networks will be dissimilar in structure. This expectation was met as the degree to which RFs enhance rather than hamper each other was significantly higher in the group of adolescents without CA (compared to the group of adolescents with CA). Those results suggest that translational research could explore whether intervening on negative RF-RF interrelation, so that they turn positive and RFs can enhance each other, may enhance the protective system and eventually result in a lower risk for subsequent mental health problems.

In Chapter 4, I took the research question of how RFs are interconnected a step further and tried to unravel RF changes during the vulnerable period between early and later adolescence. To this end, I estimated RF networks for the adolescents at age 14 as well as at age 17. I then examined whether RFs change between early (age 14) and later (age 17) adolescence, through investigating (a) RF interrelations, (b) RF mean levels, and (c) the way RFs are interrelated with distress (directly and/or indirectly via other RFs). Importantly, I again explicitly examined whether RFs change differentially in groups of adolescents with (n = 631) and without CA (n = 499). In Chapter 4 the sample (N = 1130) differed slightly from the sample in Chapter 3 (N = 1139), even though I used multiple imputation methods to reduce attrition bias between age 14 and 17 as much as possible. As in Chapter 3, at age 14 the RF network was less positively connected in the group of adolescents with CA, suggesting that RFs are less likely to enhance each other than in the group of adolescents without CA. Interestingly, this difference was no longer significant at age 17. One potential explanation for this finding may be that effects that are associated with CA exposure are predominantly strong in the short-term, but decrease over time. With regard to the RF and distress mean levels I found that the group of adolescents with CA had lower RFs and higher distress at both ages. When investigating RF mean level changes all inter-personal RFs (e.g. friendships) showed stable mean levels between age 14 and 17, and three of seven intra-personal RFs (e.g. distress tolerance) changed in a similar manner in the two groups. Thus, CA does not seem to inhibit RF changes, but seems to increase the risk of persistently lower RFs. Thirdly, I examined RF-distress pathways, which did not differ in strength between the two group. At first glance this seems to suggest that RFs have a similarly protective strength in groups of adolescents with and without CA. Yet, as RF mean levels are lower and distress is higher, RF-distress pathways may overall be less advantageous in the group of adolescents with CA. Lastly, the finding that most RF interrelations and RF-distress pathways were stable between age 14 and 17 may help explain why exposure to CA is frequently found to have a lasting effect on mental health which may decrease over time but may not wane entirely.

After having examined the protective system and the naturalistic changes of the RFs, in Chapter 5, I aimed to shed light on the predictive value with which RFs reduce mental distress. Accordingly, I tried to disentangle, (a) to what degree RFs at age 14 can explain distress levels three years later, (b) which RFs are the best indicators for distress levels three years later, and (c) with what accuracy RFs can predict distress levels three years later. The results suggest that age-14 RFs explain a similar amount of variance in age-17 distress as age-14 distress can explain. Moreover, when used together RFs had a higher relative importance than age-14 distress for explaining age-17 distress. Furthermore, the results showed that high self-esteem and low brooding RFs revealed significance in models including all ten tested RFs and had the highest relative importance. Regarding the prediction accuracy

I found that RFs and age-14 distress are similarly accurate in predicting age-17 distress. The prediction accuracy was low and highly unsatisfactory when predicting continuous distress scores. However, when predicting cruder, ordinal ("low", "moderate" and "high") distress classes the accuracy was more acceptable. As such, both RFs and distress at age 14 (as well as their combination) are able to predict for about two-in-three adolescents whether they have low, moderate or high distress three years later. Crucially, the accuracy remained similar when only self-esteem, brooding and gender were used as predictors instead of all 10 RFs, gender and CA. As assessing brooding and self-esteem is time-efficient and can be strength-focussed, those RFs may be particularly promising for risk-detection and mental health screening in stigma sensitive settings (e.g. in school-based settings).

I then, in Chapter 6, studied the effects which best describe how RFs mitigate the relationship between CA and subsequent mental health. To this end, I examined whether RFs function predominantly as mediators and/ or as moderators of the deleterious relationship between CA and mental health problems. Mediation, or "resource", factors may disrupt the deleterious effect of CA on mental health, through improving or stabilizing mental health. Moderation, or "protective", factors reduce mental health problems more in adolescents with than in adolescents without CA. I found that enhancing self-esteem, low brooding, and low aggression may be promising when the aim is to disrupt or break the chain between CA and subsequent mental health problems (i.e. mediation effects). My research seemed to provide predominant evidence for (cumulative) mediation and at best marginal support for moderation effects (i.e. friendship support, family cohesion and distress tolerance revealed effects that approached but did not reach significance). Moreover, my findings revealed that for settings in which we cannot assess adversity (e.g. due to ethical hurdles, such as a missing infrastructure for risk assessments), one may want to focus on brooding, as low brooding seemed to reduce subsequent mental health problems regardless of a history of CA. Therefore, translational research may be advised to match the purpose of the studied intervention closely with the specific type of the RF effect of interest.

Finally, for Chapter 7, I set up a cohort study with exams as a naturally occurring stressor, to shed light on the nature and dynamics of RFs over the time span from before to after a stressor. Such knowledge is concerningly scarce, but seems highly relevant to eventually be able to leverage the RFs at the right time (e.g. prevention vs treatment). More specifically, I studied self-esteem, brooding and mental distress in 451 Cambridge medical students before, during and after their end-of-the-year exams. I focussed on low brooding and high self-esteem, as those two seemed to be the most important RFs in the previous chapters. I then investigated (a) whether brooding and self-esteem change from before to after exams, (b) whether they co-evolve with mental distress (i.e. mutual change), and (c) whether they mitigate increase in mental distress during exams, as well as foster recovery of mental distress after exams. While brooding did not change over time, self-esteem was higher before than during the exams, and higher after the exams than beforehand. Brooding and mental distress predicted change in each other mutualistically from before to during the exams (not for the other time-point combinations). There was no mutualistic relationship between self-esteem and mental distress, rather, self-esteem seemed to have a predominant effect on change in mental distress. Both high self-esteem and low brooding before exams mitigated increase in mental distress during exams, which suggests that both RFs are potential prevention targets. Moreover, high self-esteem during exams predicted more

**BOX 1: THE 15 KEY FINDINGS OF MY DOCTORAL RESEARCH**

1. There is a cascade of evidence on single RFs on the intra-personal and the family level, less so for the community level. Moreover, there is a high variability between studies, little evidence for replicability, and little knowledge on RFs being tested in conjunction.
2. Adolescents with a CA history have on average lower RFs and higher distress than adolescents without a CA history.
3. RFs that change between early and later adolescence seem to change in a similar manner in adolescents with and without CA. Thus, a CA history does not seem to inhibit RF changes, but to increase the risk of persistently lower RFs.
4. RFs do not seem to function in isolation, but to function as an interacting protective system with structural differences between groups of adolescents with and without CA. Specifically, the RF network for adolescents with CA seems less positively connected, suggesting that RFs are less likely to enhance each other in this group.
5. The RF system seems to differ between the two groups particularly during early and no longer during later adolescence, suggesting that effects that are associated with CA exposure are predominantly proximal.
6. RF-distress pathways do not seem to differ in strength between adolescents with and without CA exposure, but given that RF mean levels are lower and distress is higher, RF-distress pathways may overall be less advantageous in groups of adolescents with CA exposure.
7. RF interrelations and RF-distress pathways appear to be stable between early and later adolescence, suggesting that although effects associated with CA may be most pronounced proximally after the exposure, some effects do not seem to wane over the course of adolescence.
8. Age-14 RFs explain age-17 distress similarly well as age-14 distress can explain age-17 distress, substantiating the importance of the explanatory value of RFs for subsequent mental health problems.
9. RFs and age-14 distress predict for about two-in-three adolescents whether they have low, moderate or high distress three years later. Crucially, RFs appear to predict subsequent distress similarly well as distress can predict itself.
10. Brooding and self-esteem are comparably predictive as all 10 RFs together. As their assessment can be time-efficient and strength-focussed, they seem particularly promising for risk-detection and mental health screening.
11. Most of the 10 tested RFs function as mediators on the pathway between CA and mental distress. Low brooding, high self-esteem, and low aggression seem to be most robust and may be promising intervention targets for disrupting the chain between CA and mental distress.
12. There is very little support for moderation effects. At best there is marginal support for distress tolerance, family cohesion, and friendships. Thus, it remains uncertain and needs to be re-examined whether those three RFs could be targets for improving mental health particularly in adolescents with CA.
13. In settings in which we cannot assess adversity (e.g. due to ethical precautions in schools), low brooding may be the most promising intervention target, as it reduces subsequent mental health problems regardless of a history of CA, as well as after controlling for the effects of the other RFs.
14. While brooding levels seem to stay rather stable regardless of being exposed to a stressor (here a stress-inducing exam period), self-esteem seems to be highest during times of low stress, and lowest during times of high stress. Hence, different RFs appear to be differentially impacted by a stressor.
15. While both high self-esteem and low brooding before exams mitigate increase in mental distress during the stress-inducing exam period, only self-esteem during exams fosters recovery of mental distress after exams. Hence, both RFs may be fruitful prevention targets, and particularly self-esteem may be a fruitful target for interventions at times of stress.

decrease in mental distress after exams, suggesting that self-esteem additionally fosters recovery of distress and may therefore be a potential treatment target at times of stress. All derived conjectures should of course be tested in translational research, and further observational research is needed to explore inter-personal RFs.

Overall, the results seem to shed promising light onto potentially time-efficient and strength-based RF targets (see Box 1 for the key findings), which eventually may inform mental health screening, psychoeducation, prevention, treatment, as well as transdiagnostic mental health and resilience theory. Yet, before I discuss those topics in more detail, I shall in the next section draw some attention to the limitations, the strengths, the generalizability and the replicability of my research.

## 8.2 DISADVANTAGES, ADVANTAGES, GENERALISABILITY AND REPLICABILITY

Limitations that are (content-wise) specific to the CA, the mental distress, and the RF constructs shall be discussed in their respective sub-section (see 8.3 – 8.6). Below, I shall discuss general limitations of my research, particularly those that were irremediable.

### 8.2.1 Measurement and Computation

Some of the RFs used in Chapters 3 to 6 were assessed with subscales which were initially not necessarily set out to measure a resilience-enhancing factor (e.g. low brooding) and some had psychometric limitations (e.g. did not meet tau-equivalence or needed correlated error variances). For this reason, I opted to factor analyse all RFs, to ensure that as much error as possible is removed from the RFs. In Chapter 7, this was less of a problem as I carefully selected the RF subscales based on their reliability (mainly internal consistency) and validity (mainly face validity; see also Fritz, Stochl, Kievit, et al., 2020). While the CA variable (used in Chapters 3 to 6) had a good discriminant validity (Dunn et al., 2011), the CA concept was limited in its temporal precedence, as it was measured at the same time point as the RFs (i.e. at age 14). Yet, as the RFs assess concurrent protective levels at age 14, and CA was assessed as a retrospective measure for exposure up to the age of 14, the temporal precedence was most likely applicable. Moreover, CA was measured via caregiver report and can therefore not directly be biased by the adolescents' emotional states. For the mental distress index the major limitation may be that I used a rather simple factor analytic structure. Technically, I could have applied a higher-order factor model (e.g. bifactor model) or a hierarchical factor model. In Chapter 3, where I cross-sectionally looked at age 14, I used a bifactor model (Brodbeck et al., 2011), which revealed highly similar results as a simple one factor model. Based on three reasons I chose to not apply a complex factor structure, but to use a simple one-factor structure for Chapters 4 to 6. Firstly, I was not interested in identifying sub-factors (e.g. lower order factors) and was in contrast rather interested in explaining as much variance as possible that contributes to latent mental distress. Second, I used longitudinal, ordinal factor models as our data was ordered categorical and I was interested in comparing two time points. Longitudinal, ordinal factor models are rather cutting-edge and there is still discussion with regard to the best possible approach to impose and assess invariance in those models.

Hence, I opted for the simpler but feasible factor model. Third, I decided to impute longitudinal data for Chapters 4 to 6. Fitting the longitudinal, ordinal factor model on the imputed data sets required a vast amount of computational power, which reached the limits of the available computational power and may potentially not have been possible with an even more complex model. Yet, given that the mental distress model did consider the ordered categorical and the longitudinal nature of the data, while being based on imputed data, I would rather consider the mental distress index as an advantage than as a limitation of this research.

### 8.2.2 Assessment, Covariates and Confounders

There are several limitations with regard to variables that could not be taken into account. Chapters 3 to 6 contained secondary data analyses based on an existing longitudinal dataset. The use of existing data was necessary to get an adequate sample size and length of follow-up, but had the disadvantage that I could only include those RFs that were measured in this adolescent cohort. Therefore, I was only able to study 10 of the 20 RFs that were identified in my systematic review (see Chapters 2 to 6). Moreover, the systematic review operated with quite a restricted range of inclusion criteria which was necessary to keep the literature scope manageable, but must inevitably have led to the exclusion of important additional RFs. Ergo, although this doctoral research tries to be holistic, it is at best extensive. There are also potential covariates and confounders that I have chosen not to take into account, such as psychotherapeutic and psychopharmacological treatment, as well as socio-economic status. I decided not to control for those variables for three reasons. First, I was interested in a naturalistic community setting to get the most plausible, population-representative estimates of the effects of RFs. In other words, I was interested in detecting resilience promoting effects in the general population, in which some people do and others do not seek psychopharmacological or psychotherapeutic treatment. Second, data on received treatment was very limited and data on socio-economic status was extrapolated from the adolescents' postcode, limiting the accuracy and sensitivity of such information notably. Third, examining those variables would likely have been most useful when disentangling their effects rather than just controlling for them. For example, one could opt to find out whether the effects of CA on mental distress are less pronounced in adolescents who have (at least at times) received psychotherapeutic and/or psychopharmacological treatment. Similarly, one could test whether specific RFs are more helpful for adolescents who grew up in socio-economically disadvantaged circumstances. Both research aims are – beyond all question – highly important research aims, but would come with an entirely additional set of research questions, which would clearly have been out of the scope of this thesis.

### 8.2.3 Design

Except for Chapter 7, my doctoral research did only make use of observational designs, not experimental designs. The advantage of the cohort study used for Chapters 3 to 6 is that the adolescent sample is rather large (N = ~1130), rather population representative (except for SES), contains two time points and is naturalistic. The advantage of the cohort study in Chapter 7 is that it contains a naturally occurring (within-subject) stress manipulation, in the form of exam stress. A disadvantage of the study

in Chapter 7 is that the sample is not representative of the general population (i.e. only medical students) and is relatively small (N = ~451). A disadvantage of both studies is that all variables are based on self-report (with the exception of CA and two of the RFs, namely distress tolerance and expressive suppression, which were in Chapters 3 to 6 assessed via parent report). Hence, my conclusions rely on the assumption that adolescents (and young adults) can sufficiently reflect on their own resilience promoting skills and resources, and mental health levels. Although I was here interested in naturalistic and observational concepts, rather than in biological underpinnings, it would have been advantageous to have data from multiple reporters, such as caregivers, teachers, and officiaries (e.g. general practitioners or social services).

### 8.2.4 Generalisability and Causality

With regard to generalisability the most important limitation is that the ROOTS cohort sample (Chapters 3 to 6) had a higher SES than the overall UK population (Goodyer et al., 2010), and the RESIST cohort study (Chapter 7) solely included Cambridge medical students, also having on average a higher SES compared to the general population. Hence, my results are foremostly translatable to an above average SES population, potentially also outside the UK, but it may not (smoothly) translate to low-SES populations, and therefore likely not to lower income countries. Moreover, in both cohort studies only a minority of the approached young people agreed to take part, limiting the generalizability further. The probably most important advantage regarding generalizability is that hardly any exclusion criteria were used. Crucially, my research demonstrates cross-sectional as well as longitudinal relationships among RFs and mental distress and can thus not directly speak to causality. Yet, the longitudinal components shed some cautious light on temporal relationships and therefore satisfy at least one of the Bradford Hill criteria for establishing causality (Lucas & McMichael, 2005). More specifically, we cannot be certain as yet that enhancing RFs will indeed result in better mental health, which thus needs to be further tested with experimentally-controlled intervention research (e.g. with randomized controlled trials).

### 8.2.5 Sensitivity and Robustness

I either carefully imputed missing data or used estimation algorithms that can handle missing data (e.g. the full-information maximum likelihood estimator used in Chapter 7). Therefore, I believe that the analyses conducted throughout my doctoral research are computed with as much power as possible and reduce attrition bias as much as is statistically possible, enhancing the robustness of the results and thus the conclusions. For completeness, I also present results based on non-imputed data (i.e. complete cases analyses) for Chapters 4 to 6, see online Supplements I to III. Along those lines, I have aimed to further enhance the stability and accuracy of the analyses by conducting a multitude of sensitivity and robustness analyses. For example, for many models I have checked the accuracy of the model parameters through bootstrap analyses (Chapters 3 to 6). I also aimed (a) to correct for multiple testing when performing analyses with single RFs or multiple outcome variables (e.g. Chapter 3, 4 or 6), (b) to check and meet (or if not possible relax) analysis assumptions (all chapters), (c) to check and meet (or if not possible at least approach) invariance assumptions (Chapters 4 to 7), (d) to provide absolute and relative variance (e.g. Chapter 5), as well as (e) to report direction, magnitude, and

confidence intervals when suitable (e.g. Chapters 3 to 7). For analyses with a main focus on prediction (i.e. Chapter 5) I aimed to compute the analyses with both linear and categorical outcome variables, to keep my research comparable to other prediction studies (which are mainly conducted with categorical outcome variables), while also aiming for statistical precision (which is higher in linear models). Moreover, as research has indicated that males have on average less mental health problems (after adversity) than females (Bonanno et al., 2011), I provide for some chapters not only subgroup analyses for CA (i.e. Chapters 3 to 6), but I also report gender effects (i.e. Chapter 5 and 6). I controlled for gender whenever the power was high enough and a potential difference was to be expected from the literature. Yet, future research is necessary to explore gender effects more thoroughly. Overall, I have tried to keep the applied methods as state-of-the-art and robust as possible, to improve accuracy and replicability of the findings. As “all models are wrong but some are useful” (p. 2 in Box, 1979), I hope that mine belong, albeit being far from perfect, to the useful ones.

### 8.2.6 Replicability

To increase accessibility, reproducibility and perhaps even replicability, I have not only tried to thoroughly describe the samples and the methods, but I also share my analysis scripts (see https://osf.io/cqf87/) and if possible the data (see https://doi.org/10.17863/CAM.[insert-ID]; Chapter 3 ID = 20806, Chapter 4 ID = 36708, Chapter 5 and 6 ID = 46642, Chapter 7 ID = 57030). Generally, I believe that many of my findings – as I will discuss in more detail below – are fairly in line with previous literature. More importantly however, I believe that my findings notably advance the current knowledge. Not only does my research extend many previous studies due to being based on a pre-registered, systematic literature review, but my research also operates from a holistic and potentially more ecologically valid stance. Therefore, the acquired knowledge on RFs offers a preliminary, but solid, basis to inform translational research on risk detection and (strength-based) interventions, which I will outline in detail in section 8.7. That said, despite me aiming to the best of my ability to shape an as accurate, robust, and reliable picture of the findings as possible, the reasoning and interpretations that seem correct to me today may at some point turn out to be mistaken or only half the story. Therefore, all “possible answers suggested here are examples, not conclusions; some will turn out to be wrong” (p. xii in Nesse, 2019) and all should be replicated and tested in translational research before they can soundly inform intervention, healthcare providers and policy makers.

## 8.3 HOW MY RESEARCH CAN INFORM KNOWLEDGE ON THE CONCEPT OF CHILDHOOD ADVERSITY (CA)

My research has shown that CA seems on average to be associated with lower levels of RFs and with more mental distress. Importantly, CA does not seem to inhibit the change of RFs during adolescence, but seems to be related to consistently lower levels of RFs and higher levels of distress. Moreover, while RF interrelations of adolescents with CA seemed only to be disadvantaged proximally after CA, the protective pathways between the RFs and distress seemed to be disadvantaged both during early

and later adolescence. This suggests that not all effects associated with CA wane during the course of adolescence. Hence, adverse experiences before the age of 14 seem to have a critical relationship with both RFs and mental health problems, though the degree of the deleterious impact CA seems to have on RFs and mental health problems may decrease over time and needs further investigation.

Importantly, besides the aforementioned general limitations of my research, there are several limitations that are specific to the CA index used here: (a) we had no self-report that could have corroborated the caregiver report (Steptoe et al., 2019), (b) the assessment was solely done retrospectively (Steptoe et al., 2019), (c) there was little assessment regarding community adversity (e.g. a violent neighbourhood) or social inequalities (e.g. racial discrimination, food insecurity; McEwen & Gregerson, 2019), (d) my results may not translate directly to those studies that assessed CA also later than age 14 (e.g. until age 18; Felitti et al., 1998) and (e) in contrast to several adverse childhood experiences (ACE) studies, which generally use a sum score for all types of experienced CAs, I used a binary index for “any vs no” CA (Felitti et al., 1998). Moreover, our CA measure differed in some additional ways from rather concise ACE measures. Firstly, in most of my chapters (i.e. Chapters 3 to 6) adverse experiences were assessed with a semi-structured interview rather than with a questionnaire. Secondly, in comparison to traditional ACE measures, our used interview included additional adverse experiences, such as a family member with an impairing chronic or life-threatening physical illness.

While an aggregated adversity index as used in my thesis is on the one hand indeed a simplification, it also comes with advantages. The justification for treating CA as an aggregated index, rather than as separate forms of traumatic events or severe stress, stems from a solid body of research showing that CAs often co-occur and that most forms are strongly associated with subsequent mental health problems (Greif Green et al., 2010; Kessler et al., 2010; McLaughlin, 2016; McLaughlin et al., 2012). Our CA measure identified that 56% of all adolescents were exposed to at least a moderate level of adverse experiences, which aligns closely with many other population representative studies (McLaughlin, 2016). Thus, the simple binary CA index can be considered advantageous for my research, as it simplified the research design, increased analytic power, and made results more easily digestible than a cumulative count index would have done.

The perhaps most relevant limitation of the CA index used here may be that the assessment was exclusively based on retrospective caregiver (usually maternal) report. As well as recall bias, there is a possibility of under-reporting if the caregiver felt (in part) responsible for the adversity. Future research is clearly needed that tests CA effects in a prospective design, such as a birth cohort study. This would also enable taking baseline levels of RFs and mental health before the exposure to CA into account. There is for example the possibility that pre-exposure RF levels contribute to the exposure risk and therefore may even at this early stage be valuable targets for identification and prevention (Danese, 2019). Such a design may also enhance the detection of sensitive periods during which exposure to CA has a particularly detrimental effect (McLaughlin, 2016). For example, research in institutionalized children has found that the establishment of secure attachment is predominantly achievable before the age of two (McLaughlin, 2016). Ideally, it would have been best to have both a prospective and a retrospective index, as those indices have been shown to only overlap slightly and to lead to different

insights (Danese, 2019). For example, Danese (2019) showed that 52% of the people did not report maltreatment events retrospectively, which were observed prospectively. Moreover, 56% of the people reported maltreatment events retrospectively, which were not observed prospectively. Along those lines, it would also have been advantageous, and would be recommendable for future research, to have multiple reporters, such as both the caregiver's as well as the young adolescent's report on the adverse experience (at least if recall of the event is possible). As discussed in the introduction, cutting-edge research suggests that subjective, self-reported CA is a much stronger predictor for subsequent mental health problems than more objectively reported CA (Danese & Widom, 2020). Hence, speculatively, it may be that our CA variable, being derived from caregiver report, may have resulted in a smaller effect than a comparable CA variable derived from adolescent report would have yielded. Hence, if at all, our detected CA effects are more likely to err on the conservative side.

Besides the fact that my doctoral research cannot answer whether the disadvantaged RF and mental distress levels were already present before the exposure to CA (Danese, 2019), my research does not speak to (a) whether a different exposure timing has a different effect on the type and the strength of mental health problems (Steptoe et al., 2019), (b) whether different types of CA have a different effect on the type and the strength of mental health problems (Merrick et al., 2019; Steptoe et al., 2019), (c) whether a different exposure timing has a different effect on the protective effect of RFs, and (d) whether different types of CA have a different effect on the protective effect of RFs (Merrick et al., 2019; Steptoe et al., 2019). Clearly, the latter four endeavours were far beyond the scope of my thesis and would not only have required a bigger sample size, but ideally also narrower assessment windows (e.g. see my suggestion in Box 2). However, given that my doctoral findings underpin that CA has not only a detrimental relationship with mental health but also with RFs (which may well wane somewhat over the course of adolescence), I believe that it would be very sensible for future research to build upon my findings and explore potential CA timing and CA type effects. McLaughlin (2016) for example suggests that it may be worthwhile to split the CA index into deprivation and threat, to gain knowledge on whether those two dimensions have different pathways with RFs and mental health problems (McLaughlin, 2016). A related topic for future research may be to study the suitability of different facets of CA (e.g. severity, frequency and duration). For example, our lab has recently shown that CA severity and frequency may be better indicators than only the presence (set off against the absence) of CA (Schlechter et al., 2019). Research assessing CA severity would then also be able to explore stress inoculation effects. The stress inoculation theory suggests that high as well as very low CA has a negative impact on mental health, whereas a moderate level of CA may have an immunization effect and therefore eventually a positive impact on mental health (Seery et al., 2010). However, the U-shaped CA idea may be restricted to specific types of adversity. Seery and colleagues (2010), who spearheaded inoculation research, explain that for example unemployment may not prepare for subsequently losing employment and financial security again. Hence, research on the U-shaped CA hypothesis is high time, but was unsuitable for the current thesis.

From the cascade of research that has been conducted on CA, it is crystal clear that there is a high need for more congruency in the CA definition (McLaughlin, 2016). I would therefore plead for a prospective assessment, and if that is not possible, to have a retrospective measure ideally completed

by multiple informants (e.g. the adolescent, the parent, and the teacher). Moreover, besides taking severity, frequency, duration and the onset into account, I would suggest adding a component of clinical significance to the concept (see my measurement suggestion in Box 2). This could for example be based on the suggestion from McLaughlin (2016) that CA has to go together with a notable divergence of a reasonable living situation, or a notable alteration of the anticipated living situation, and should thus entail active adjustment by the child or adolescent. Such a criterion would then allow to filter out natural hassles, such as minor parental upheavals.

Congruency may however not only be advantageous in terms of measurement type (retrospective versus prospective), assessment facet (e.g. severity versus frequency), and reporting type (e.g. self-report versus parent report), but also in terms of content range of CAs. For example, while in my thesis Chapter 2 captured quite a range of environmental adversities (e.g. rocket attacks), Chapters 3 to 6 mainly captured family-related adversity. I provide a carefully selected assortment of potentially important CAs in Box 2. As mentioned in the introduction, a different but related topic is that researchers disagree with regard to the question whether poverty should fall under the CA umbrella or should be a factor of its own (Steptoe et al., 2019). One reason is that not necessarily poverty itself but that intermediate factors may increase the risk of subsequent mental health problems. For example, a child may not receive sufficient care (i.e. may be neglected) not because the parents do not want to care for the child, but because of the heavy workload and the long working hours the parents have to handle to overcome the poverty they are facing. From whichever perspective reasoned, my research cannot directly speak to poverty, which is definitely a highly important topic for future research.

**BOX 2: A DIMENSONAL SUGGESTION FOR MEASURING ADVERSE CHILDHOOD EXPERIENCES**

Content

- Exposure to adversity can either be the result of a single traumatic event (e.g. death of a significant other), or the result of several severely stressful events (e.g. parental mental illness with significant impact on the family life; McLaughlin, 2016). Including:
    1. Sexual abuse (e.g. rape, molestation)
    2. Physical abuse (e.g. victim of violence other than sexual, including spanking)
    3. Emotional abuse (e.g. terrorising, humiliating other than by peers)
    4. Peer bullying (e.g. terrorising, humiliating by peers)
    5. Emotional neglect (e.g. no one who looks/ looked after you, made you feel important)
    6. Physical neglect (e.g. inadequate food and clothing)
    7. Witnessing violence within the family (e.g. witnessing intimate partner violence)
    8. Witnessing violence outside of the family (e.g. stabbings or shootings)
    9. Growing up in a violent environment (e.g. due to wars, terrorism, political conflicts, ethnic conflicts, torture, or gangs)
    10. Severe criminal activities of a parents, siblings, or significant other (severe enough to cause problems)
    11. Severe gambling, or alcohol, or drug use of a parent, sibling, or significant other (severe enough to cause problems)

12. A parent, sibling, or significant other with a mental illness and/ or a suicide attempt
13. Stressful family transition (e.g. a major upheaval between the parents, divorce, separation)
14. Separation from parent(s) for more than 1 year (e.g. because of prison time)
15. Having been raised by someone else than the biological parent(s) (e.g. foster care)
16. Death of a very close friend or family member (except for grandparents, unless they are/ were the primary caregivers)
17. A parent, sibling, or significant other having been/ being seriously ill or injured (e.g. a chronic or life-threatening disease)
18. Having been/ being seriously ill or injured (e.g. a chronic or life-threatening disease)
19. Exposure to/ having witnessed a traumatic accident (e.g. car crash, even if it did not result in life-threatening injuries)
20. Financial difficulties within the family (e.g. unwanted unemployment for more than 1 year, poverty)
21. Destruction of home or close environment (e.g. due to war or due to a nature catastrophe such as a flood, a hurricane, or an avalanche)
22. Growing up in an unhealthy environment (e.g. environmental pollutants, overcrowded housing, homelessness)
23. Or anything else that was experienced as traumatic event or the result of several severely stressful events that had a significant impact and is not covered by the above
    *items taken from (Afifi et al., 2020; Felitti et al., 1998; McEwen & Gregerson, 2019; Schlechter et al., 2019; World Health Organization (WHO), 2018)

- Clinical significance: CA should correspond to a notable divergence of a reasonable living situation, or a notable alteration of the anticipated living situation, and should thus entail active adjustment by the child or adolescent (McLaughlin, 2016).

Components

➔ Ideally all components should be assessed

- Severity: how traumatic was the event/ series of events?
- Frequency: how often did the event or the series of events happen?
- Duration/ Chronicity: how long did the event/ series of events last?
- Onset: how old was the child/ adolescent when the event/ series of events started?
    1. infant/ toddler (age 0 to 3)
    2. early childhood (age 4 to 6)
    3. late childhood (age 7 to 10)
    4. preadolescence (age 11 to 13)
    5. adolescence (age 14 to 18)

Measurement

➔ Ideally multiple types should be used

- prospective interview
- prospective questionnaire
- retrospective interview
- retrospective questionnaire
- official records

Informant

➔ Ideally multiple informants should be recruited

- the child/adolescent
- a caregiver
- a teacher
- an officiary (e.g. general practitioner or social services)

A last remark for the concept of CA in relation to mental health promotion is a prevention idea that has been discussed by Patel and colleagues (2007). They suggest that, as many adverse experiences happen within the family (e.g. neglect), it may be worth trying to educate parents in parenting and thereby reducing the occurrence of CA in the first place. Hence, prevention of CA rather than prevention of mental health problems following CA (Patel et al., 2007). There is preliminary evidence that home visitation programs could deliver such prevention (Felitti et al., 1998; Jones, Merrick, & Houry, 2020). A highly interesting idea, however future research needs to shed more light on how implementable and effective such prevention strategies are. Moreover, such a type of prevention would only be relevant for family related adversity and would not prevent exposure unrelated to the family (e.g. a car crash; Patel et al., 2007). Although CA is generally a topic that needs to be dealt with on the societal level, it seems important that clinicians (including mental health but also paediatric practitioners) are trained in assessing, detecting and dealing with their patients' exposure to CAs (Jones et al., 2020). More detailed assessment suggestions can be found in Box 2.

## 8.4 HOW MY RESEARCH CAN INFORM KNOWLEDGE ON THE CONCEPT OF MENTAL HEALTH

With regard to mental health my research has, in line with past research, shown that mental health is negatively impacted by CA, but positively impacted by RFs. Moreover, in my research mental health could best be described as a continuous latent factor, but based on this factor it was possible to identify severity subgroups (i.e. low/ mild [n = 623], moderate [n = 390], and high [n = 117] mental distress) using a data-driven clustering approach. Interestingly, low, moderate and high mental distress classes could similarly well be predicted by RFs and prior mental distress. A notable restriction of such a transdiagnostic mental health conceptualization comes from the fact that we cannot draw conclusions about criterion-based mental illness (as e.g. with the DSM, American Psychiatric Association, 2013; or the ICD, International Statistical Classification of Diseases and Related Health Problems, World Health Organization, 2010), but just about mental distress ranging from low to high. Statistically and theoretically this seems advantageous and appears to reflect the nature of mental health well. Statistically it seems advantageous, as it minimizes the loss of information that occurs through categorizing, and thereby comes with more statistical power (Senn, 2018). Theoretically it seems advantageous, as it for example does not neglect those people who suffer from significant mental distress, but do not qualify for a mental illness, as they do not have the correct symptom count or symptom constellation (Dalgleish, Black, Johnston, & Bevan, 2020). A related advantage is that transdiagnostic continua seem to be better suited to take into account a broad range of bio-psycho-social determinants and symptoms, which are difficult to be included and captured in binary "presence" vs "absence" diagnostics. Dalgleish and colleagues (2020; p. 181) state that a mental health problem "prototypically emerges from an interplay between myriad biological, behavioural, psychosocial, and cultural processes that do not respect established diagnostic boundaries, where the interactions are multifarious, and modulated by an individual's lifelong experiences of the world.". Furthermore, research on the phenomenon of multi-finality has clearly shown that the same adverse experience, such as

abuse, can lead to various different disorders (Nolen-Hoeksema & Watkins, 2011). In other words, there is little specificity in factors contributing to the aetiology of different diagnoses (Borsboom, Epskamp, Kievit, Cramer, & Schmittmann, 2011; Dalgleish et al., 2020).

Importantly, transdiagnostic indices elegantly capture comorbidity, not only through the symptom breadth but also in terms of mental health severity. This is highly important as, throughout the life course, more than one quarter of all people suffer from more than one mental health disorder (Kessler et al., 2005; Nolen-Hoeksema & Watkins, 2011), and their diagnoses have been found to fluctuate between disorders (Nolen-Hoeksema & Watkins, 2011). According to Chu, Temkin and Toffey (2018; p. 3) “the high rates of comorbidity . . . seen in adult populations are even higher in children and adolescents, where both within-class (e.g., multiple anxiety diagnoses) and across-class comorbidity (e.g., diagnosis of anxiety and conduct disorder) make comorbidity the rule rather than the exception.”. A transdiagnostic index as used here overcomes not only comorbidity but also symptom heterogeneity, as it simply considers an extensive symptom breadth. For example, some adolescents with internalizing symptoms sleep too much and others too little, which are opposing symptoms, but both contribute to clinical severity (Borsboom, Epskamp, et al., 2011). Along those lines, dimensional transdiagnostic factors have been found to have a better validity and reliability than binary diagnostic approaches (Dalgleish et al., 2020).

That said, the transdiagnostic index used here is not fully transdiagnostic, as it only covers internalizing but not other mental health problems, such as externalizing or thought disorder symptoms. Of course, conceptualizing a fully transdiagnostic index, covering internalizing, externalizing and thought disorder symptoms would be most inclusive. Yet, focussing on internalizing is already advantageous in itself, as it has been shown to capture sequential comorbidity, to hold irrespective of gender (or sexual orientation), to replicate across cultures, and to be temporally stable (while diagnoses tend to change over the life course; Krueger & Eaton, 2015). Hence, there is a good chance that the RFs which I have investigated have a rather universal effect on people with internalizing related disorders, including those with comorbid symptom presentations of other disorders not captured in my index. Particularly because comorbidity is so high, targeting transdiagnostic RFs is theoretically likely to not only act upon concurrent symptoms, but to also prevent potential other future symptomatology (Nolen-Hoeksema & Watkins, 2011). Unsurprisingly, transdiagnostic oriented interventions for internalizing disorders have been found to be at least as good as diagnosis-based comparison interventions (Dalgleish et al., 2020).

A limitation of transdiagnostic indices is that without validated “cut-offs”, it seems challenging to translate transdiagnostic continua for diagnostic and clinical settings. The classes which I added on top of the used transdiagnostic mental health index could be seen as compromise between considering the continuous nature of mental health problems, while at the same time allowing for a cruder but more easily graspable severity classification. However, the probably most crucial limitation is that we cannot disentangle effects that differ between specific mental health problems (e.g. anxiety vs depression symptoms). Although such a research endeavour was not set out for my research, and is not only beyond the scope of my thesis but would most likely also require a bigger sample size, it would be

interesting for future research to disentangle whether given RFs lend themselves better for preventing some disorders than disorders.

All in all, due to the simplicity of transdiagnostic mental health indices, they provide an elegant solution for modelling multiple mechanisms or factors simultaneously (Chu et al., 2018). This was essential for my doctoral research, as it enabled the evaluation of multiple RFs at the same time, in relation to one simple, but comprehensive mental health indicator. In situations where it is not essential for a research question to rely on – relatively simple – continuous mental health indices or mental health classes, one may well be interested in investigating mental disorders, or comorbidity between disorders, with an overarching network model (Borsboom, Epskamp, et al., 2011). This approach has started discovering interesting symptom interactions and comorbidity characteristics (Fried & Cramer, 2017).

Another highly interesting approach would be to extend the continuous mental health concept – i.e. low to high mental health problems – with well-being. In other words, instead of only assessing whether someone has no or low mental health problems, to also assess whether this person scores high on well-being. Research has shown that mental health problems and mental well-being correlate only moderately with each other (Slade, 2010). According to the "Complete State Model of Mental Health" well-being and mental health problems are separate dimensions (see Figure 8.1), which means that someone with high mental health problems can still function well (i.e. struggling) but can also function poorly (i.e. floundering), while someone with low mental health problems can function well (i.e. flourishing) but can also function poorly (i.e. languishing; Slade, 2010). Gaining such specific knowledge may be worthwhile for informing clinics, as not only a low level of distress symptoms or the absence of

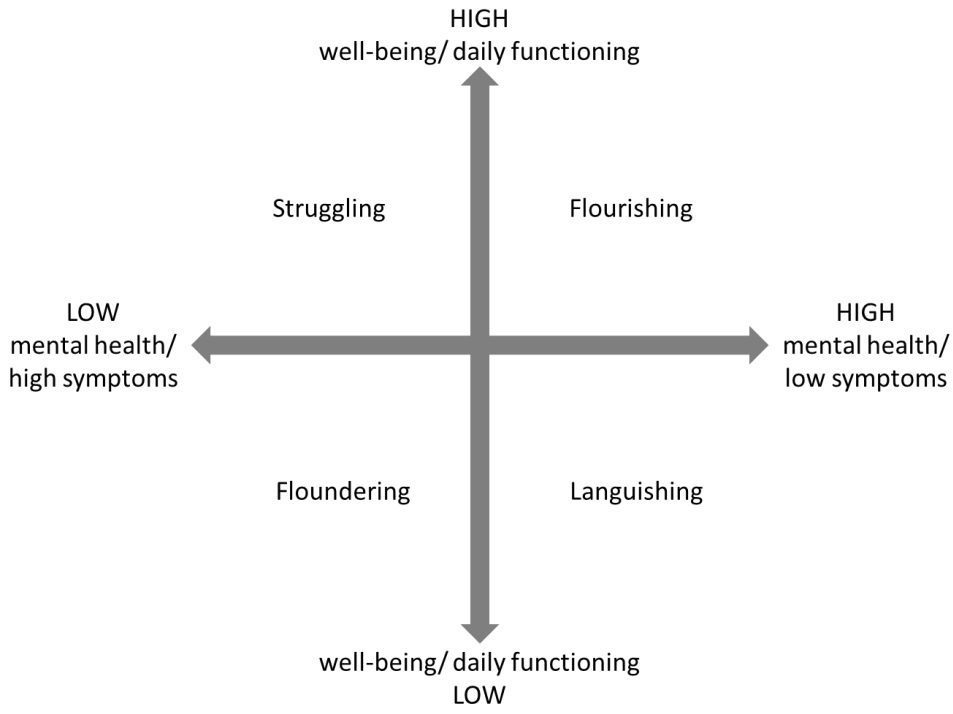



*Figure 8.1.* Depicting the orthogonal mental health (x-axis) and well-being (y-axis) dimensions of the "Complete State Model of Mental Health", adapted from Figure 1 in Slade (2010) BMC Health Services Research, 10:26; http://www.biomedcentral.com/1472-6963/10/26 [the here depicted Figure was designed manually; only the underlying concept and the four pillars struggling, flourishing, floundering and languishing were duplicated from the original article].

psychopathology, but also mental well-being contributes crucially to a healthy mental state (Slade, 2010). Moreover, such knowledge may be equally worthwhile for public mental health promotion, as both people with and without mental health problems can experience and profit from promoting mental well-being (Slade, 2010). In sum, while there are several possible improvements and extensions to the transdiagnostic mental health index used throughout my research, it already comes with multiple empirical and clinical strengths, through which it has facilitated the detection of a small number of potentially crucial RFs that help us to better predict and understand internalizing-related mental health problems.

## 8.5 HOW MY RESEARCH CAN INFORM KNOWLEDGE ON ADOLESCENT MENTAL HEALTH

As most of this research was done with adolescents and young adults, not all findings may translate to childhood, later adulthood, and old age. For example, parental support has been suggested to be less important during adolescence than during childhood (Hostinar, Johnson, & Gunnar, 2015). The adolescent years are, however, crucially relevant. The WHO (2018) for example reported that about fifty percent of all mental illnesses start emerging during late childhood and adolescence. The US National Comorbidity Survey Replication Study (Kessler et al., 2005), in which 9,282 people were assessed, found that about 75 percent of all mental disorders start to emerge during adolescence and young adulthood (Patel et al., 2007). As the adolescent time period is also known to be amenable regarding social, emotional, cognitive and behavioural functioning, targeting this age-group may be particularly successful and may foster or improve mental health before moving into the more independent and autonomic adult period. In other words, the adolescent years seem to be a particularly fruitful prevention or intervention period, as good mental health is likely to contribute to stepping stones that are set in this period (e.g. education, cognitive development, inter-personal skills, and romantic relationship experiences), which partially determine success (in those areas) in later life (Patel et al., 2007). Hence, supporting individuals in such a vulnerable life period does seem to make much sense (Patel et al., 2007).

Based on the findings discussed throughout my thesis, it seems apparent that mental health promotion should not only be focused on the adolescent as an individual, with given symptoms (e.g. anxiety or depression symptoms) and skills (e.g. self-esteem), but should also take into account the adolescent's external resources, such as family (e.g. family cohesion) and community assets (e.g. friendship support). Importantly, research on adolescent mental health indicates that there are profound differences between boys and girls. For example, girls are more prone to develop depression related symptoms, whereas boys are more prone to develop behaviour related symptoms (Patel et al., 2007). In several chapters in this thesis gender effects were controlled for (e.g. in Chapter 5 and 6). However, I did not specifically investigate gender effects in detail, as not all analyses had enough power to be split for gender, and because I felt that another layer of complexity may have complicated the results too much. Future research is strongly advised to more specifically disentangle gender effects. In sum,

although I believe that the here presented findings are highly relevant, a life course perspective and a detailed exploration of gender effects are necessary to form a complete picture of the value of RFs.

## 8.6 HOW MY RESEARCH CAN INFORM KNOWLEDGE ON THE CONCEPT OF MENTAL HEALTH RESILIENCE

With regard to mental health resilience my research has mainly revealed (a) that RFs function indeed on multiple ecological levels (e.g. on intra- and inter-personal levels), (b) that there is so far only little replication for the promotive effects of RFs, (c) that RFs function as a complex protective system, (d) that brooding and self-esteem RFs were most predictive for subsequent mental health, (e) that enhancing self-esteem, low brooding, and low aggression may be promising when the aim is to disrupt or break the chain between CA and subsequent mental health problems, and (f) that while brooding seemed to be rather stable, regardless of the stress level, self-esteem was highest during times of relatively low stress and lowest during times of relatively high stress. Importantly, as I have used a transdiagnostic mental health factor, the RFs can be understood as transdiagnostic promotive factors. Moreover, most of the studies so far only looked at single RFs, which is likely to overestimate RF effects. I, in contrast, studied the RFs as complex protective system and tested which RFs are most beneficial when a multitude of RFs are taken into account. This is clearly an advancement, but also still limited, as I could only test those RFs that were assessed in our cohorts.

Before I try to shed light on how the revealed knowledge can inform resilience theory, I shall make a – perhaps bold – suggestion. I would like to suggest for future research to re-label "*resilience*-promoting factors" as "promotive factors". Let me explain this in more detail. When I investigated single RFs, all RFs except for expressive suppression and distress tolerance were significantly associated with a lower level of subsequent mental distress in both the group of adolescents with and without CA (of note, all effects are corrected for multiple testing). Distress tolerance was only significantly negatively associated with subsequent mental distress in the group of adolescents with CA, and expressive suppression in neither of the two groups. When taking the effects of the other RFs into account, six RFs were still negatively associated with subsequent mental distress in the group of adolescents with CA (namely friendship support, family cohesion, positive self-esteem, brooding, distress tolerance, and aggression), and two in the group of adolescents without CA (negative self-esteem and brooding). Moreover, in the network models (see Chapter 4) the paths between RFs and mental distress did not differ in strength between the two groups. Hence, my research suggests that (a) most single RFs have a promotive effect on mental health in both the group of adolescents with and without CA, (b) some RFs have a promotive effect in both groups even after controlling for the other RFs, and (c) RF-distress pathways do not seem to differ in strength between the two groups. I believe that re-labelling "*resilience*-promoting factors" as "promotive factors" would ensure that it is clear that those factors can also be promotive for adolescents without a history of adversity. This is probably particularly important when we want to move forward targeting some of those factors in settings in which we cannot or do not want

to assess an adversity history, and aim to foster or improve mental health in all individuals regardless of an adversity history.

A closely related caveat, that may also be prevented by using the more general term “promotive factors”, is that people may wrongly assume that “*resilience*-promoting factors” have a higher mean level in adolescents with than in adolescents without CA. This is however clearly not the case, rather it is the exact opposite, as all RFs except for reflection had a lower mean level in the group of adolescents with CA. As the more general term “promotive factors” does not directly speak to resilience and therefore also not to adversity, the interpretation may be simpler and clearer: namely, a higher levels of promotive factors mitigates mental health problems and promotes good mental health. For consistency I will throughout the rest of my doctoral thesis stick to the term RFs.

I will now reiterate and discuss two caveats that were already mentioned in the introduction. The first one is whether resilience and risk factors are opposing sides of the same continuum. The quick, but insufficient, answer for the research in this thesis is probably that some of the investigated RFs seem indeed to be the flip side of risk factors. For example, while a high level of friendship support is known to be protective, a low level of friendship support is often understood as a risk factor (van Harmelen et al., 2016). Another example may be self-esteem. Self-esteem (or a positive self-concept) is commonly defined as RF and has been discussed as such by many of the seminal resilience researchers, including Michael Rutter, Emmy Werner, Ann Masten, and Michael Ungar (for a review see e.g. Shean, 2015; or Traub & Boynton-Jarrett, 2017). Yet, at the same time, a low level of self-esteem or self-worth is part of the DSM 5 criteria for depression ("Feelings of worthlessness”; American Psychiatric Association, 2013). Hence, whereas a high level of self-esteem may protect against low mood levels, low self-esteem is assumed to contribute to or reflect low mood. For other RFs, this apparent dichotomy is somewhat more complex. For example, consider negative and positive rumination. Low negative rumination, called brooding throughout this thesis, has been solidly found to be linked to a lower level of depression, while high negative rumination has been linked to higher levels of depression (Harding & Mezulis, 2017). Similarly, some research suggests that high positive rumination is linked to a lower level of depression, and particularly to lower levels of anhedonia, while low positive rumination is linked to higher levels of depression or anhedonia (Harding & Mezulis, 2017). Negative and positive rumination have been found to be positively related, and have been suggested to overlap in terms of affect amplification, but to neither be the same construct (i.e. the former focusses on negative and the latter on positive content), nor to operate on opposing sides of the same continuum. More specifically, it has been suggested that some people who ruminate more on negative content, also ruminate more on positive content; hence, the two very related constructs are neither the same, nor do they operate on the same continuum, rather they are similar constructs of opposing content which operate – if you want – on parallel continua (i.e. high positive as well as low negative rumination on the one end and low positive as well as high negative rumination on the other end; Harding & Mezulis, 2017). The attentive reader may wonder whether this apparent dichotomy may as well apply to self-esteem, once its content valence is taken into account (high positive self-esteem = RF and low positive self-esteem = risk factor, while high negative self-esteem = risk factor and low negative self-esteem = RF). In sum, I cautiously suggest that on the group level some RFs may operate on a continuum with

risk factors, while for others the relationship with risk seems to be more complex. Importantly, however, regardless of whether resilience and risk factors operate on the same continuum, are inversely correlated but not identical, or are positively correlated but content wise opposing, understanding the nature of RFs seems to have universal appeal as it focuses on what promotes good mental health rather than on what increases mental health problems.

The second caveat, also already introduced in the introduction, is that research on RFs comes with the danger that people may think that they have to be able to possess sufficient RFs to thrive (naturally), and that it is their own fault if they do not develop RF skills or recruit RF resources to be able to thrive (Infurna & Luthar, 2018). I want to repeat here, that the idea of this research is not to find out which RFs people naturally should possess, the idea is to find out which RFs are most advantageous and can theoretically be targeted in interventions, to eventually help more people to have a stable mental health or to recover to an acceptable level of mental health.

Besides the fact that we may have to re-think how to coin RFs properly and how to more neatly set them off from risk factors – if at all possible – there are several other important limitations to consider for future research. For example, an important question that needs to be answered by future research is whether the promotive value of RFs differs across the life course. As discussed in Chapter 4, I found no significant difference in RF-distress pathways between age 14 and 17. Thus, future research is clearly needed to disentangle over a longer time span whether periods exist during which RFs are more, or less, protective. Importantly, all RFs studied here are expected to be malleable, which may enhance the likelihood of sensitive developmental windows. In Chapter 7 I provide a preliminary insight into the differential promotive value of RFs before, during and after a stress-inducing exam period. More specifically, while both low brooding and high self-esteem before exams mitigated increase in mental distress during exams, only self-esteem during exams fostered recovery from mental distress after exams. However, Chapter 7 cannot speak to long term promotive effects of RFs after the stressor, as we only had one assessment a couple of months after the stress-inducing exam period. Moreover, Chapter 7 was limited in that it can only speak to stress and not to adversity exposure. Therefore, future research is needed to explore the effects of RFs over the time span from before to after CA (rather than to stress). For example, preliminary evidence exists that low rumination before CA is associated with less mental health problems after CA (Bonanno et al., 2011). Thus, it would be interesting for future research to explore whether a pre-CA, during-CA, or a post-CA RF is a better promotive factor for mitigating subsequent mental health problems, or whether they are similarly promising.

Another as yet unanswered question is at which ecological level (e.g. individual, family or community) one should best intervene. Obviously, my findings are observational and can thus only inform translational research, but not directly interventions. Given my results in Chapter 3 and 4, where I tested the RFs as an interacting system, it seems likely that considering both the intra-personal (e.g. self-esteem and brooding) as well as the inter-personal level (e.g. family cohesion) may be most fruitful, as both levels were highly central and may contribute to mental health resilience. Ungar and Theron (2020) recently put forward a multi-level resilience framework that suggests components ranging from biological to psychological (i.e. throughout this thesis called intra-personal), to social, to environmental levels. Hence, an important limitation of my doctoral research is that none of the RFs extends to broader

community, socioecological, or cultural contexts, such as social justice, sense of collective cohesion or efficacy, and cultural adherence (Ungar & Theron, 2020). In the systematic-review, in Chapter 2, I found that most studies that inform mental health resilience research focus on the intra-personal level. Therefore, it would be advisable for future research to also, or perhaps particularly, focus on the inter-personal, societal level, and perhaps even on the environmental and cultural levels. Along those lines it is important to note that the effectiveness of interventions targeting RFs may largely depend on access to basic resources, such as adequate food supply, safety, housing, physical well-being, and education (Bonanno et al., 2011; Ungar & Theron, 2020). Ungar and Theron suggest that: "More multidimensional and multilevel interventions also reduce concerns that a focus on resilience serves neoliberal agendas by blaming those who do not thrive for their low level of success" (p. 5 in Ungar & Theron, 2020). A related limitation is that all RFs evaluated here, are only evaluated in the context of mental health and not for example set off against educational attainment or life satisfaction (Bonanno et al., 2011; Luthar & Eisenberg, 2017). However, this was also clearly set out as aim of this research, to keep the frame realistic, and is therefore rather a constraint than a drawback.

In addition to the longitudinal nature and the contextual scope of the RFs, the nomothetic group-level approach at which RFs are studied throughout my doctoral work is limited. Although some of the RFs appeared to be promotive for the majority of the adolescents, no one RF will be protective for everyone. All RFs are likely to be dependent on the type of adversity experienced, the person's genetic make-up, the socio-cultural context and support system, the proneness for vulnerability to specific symptom clusters, the level of functioning (or impairment) in daily life, and potentially on the mental illness course and chronicity. One partial solution to this limitation would be to identify those RFs that are most independent of the person-specific factors. Another partial solution – which would likely come with valuable, additional insight – would be to conduct idiographic individual-level research, to identify those RFs that appear to be most helpful for a specific person.

A criticism for the general conceptualization of mental health resilience used throughout this thesis stems from the novelty of the underlying theory, or more specifically, the absence thereof. For example, according to Bonanno and colleagues (2011; p. 528) "defining resilience as the absence of a disorder is akin to defining health as the absence of disease (Almedom & Glandon 2007) and does little to advance our understanding of genuinely resilient outcomes". Overall, I have to admit being guilty, I did define mental health resilience as (on average) good mental health following adversity. That is, I used adversity as predictor and mental health as outcome, rather than having a combined resilience coefficient (e.g. see Ioannidis et al., 2020; Kalisch et al., 2015) or resilience classes (e.g. see Bonanno et al., 2011) as outcome. Hence, yes, my definition of mental health resilience is clearly limited. However, the main aim of this thesis was not to define or conceptualize mental health resilience in itself, but to explore the nature of those factors that may potentially promote mental health, in individuals with, but also in individuals without a history of adversity.

Last but not least, the question remains whether the here-revealed knowledge on RFs can inform (preventative) interventions. Bonanno and colleagues (2011; p. 523) state that "prophylactic programs . . . have been developed primarily on the basis of cross-sectional, retrospective studies of adjustment on the one hand and literature on risk factors in psychopathology on the other, and thus in

their current form rest on underdeveloped and potentially misleading theoretical and methodological foundations." On the other hand, Luthar and Eisenberg (2017; p. 337) have confidence that "With over six decades of research on childhood risk and resilience (Cicchetti & Curtis, 2006; Luthar, Crossman, & Small, 2015; Masten & Narayan, 2012), applied scientists are at a juncture where we can, and indeed should, distill robust findings to derive top priorities for interventions." Likely both viewpoints hold some truth. Importantly however, I believe that in contrast to many of the current research studies, my research has improved pre-existing knowledge on RFs, as it was mainly longitudinal, focused on amenable factors, was based on an empirical systematic review and thus had a sound theoretical basis, and was conducted with transdiagnostic mental health indices instead of binary disorder classifications.

While strength-based approaches may well be effective in at-risk populations, Bonanno and colleagues (2011) caution that resilience enhancing in the general population – without any particular concern for mental health problems – may potentially be an erroneous idea, as it (a) may rather shift the risk perception and not risk protection or (b) may even weaken or reverse the person's usual skills. Therefore, some precautions regarding the theory behind mental health promotion via RFs seem necessary. One potential account may be provided by the risk homeostasis theory. Risk homeostasis theory proposes that individuals constantly assess their level of risk in comparison to the related action and context, so that if the risk is too large to be tolerable they can adapt the action and/or context to reduce the risk (Bonanno et al., 2011; Wilde, 1982). Thus the idea is that RFs and risk factors are kept in balance (e.g. using a seatbelt may decrease caution and increase unsafe driving, because the person feels safer; Bonanno et al., 2011; Wilde, 1982). Hence, the question that evolves with regard to mental health is whether increasing the level of RFs will (a) indeed tackle the negative impact of risk and stress and thereby increase mental health (and/or well-being), as suggested by strength-based interventions, or will (b) increase the tolerance for risk or stress and therefore in sum not change the overall level of mental health (and/or well-being).

In Chapter 7, I tested a similar scenario. Specifically, I tested whether the relationship between protective factors and distress changes mutualistically when a person is exposed to stress. To this end I used an experimental cohort design with a natural stressor, namely high-stakes end of year exams. Firstly, my findings suggest that while brooding levels do not change significantly, self-esteem decreases during exams and increases afterwards to a higher level than prior to exams. Hence, self-esteem had the opposite trajectory to mental distress, which increased during exams and decreased afterwards to a lower level than prior to exams. For the mutualism models, several outcomes were possible, but four seemed theoretically most plausible. For brevity I will sketch them here only for the time from before to during exam stress: (a) positive homeostasis, which means that distress and the RF predict change in the respectively other positively (the higher the RFs, the higher increase in distress and the higher distress, the lower decrease in the RF) and which would align with the risk homeostasis theory, (b) negative homeostasis, which means that distress and the RF predict change in the respectively other negatively (the higher the RFs, the lower increase in distress and the higher distress, the higher decrease in the RF) which would probably align with the idea of strength-based interventions, (c) non-mutualistic strength dominance (or distress inferiority), which means that the RF predominantly predicts change in distress (the higher the RFs, the lower increase in distress, but no significant impact

of distress on the RF) which would probably also align with the idea of strength-based interventions, or (d) non-mutualistic distress dominance (or strength inferiority), which means that distress predominantly predicts change in the RF (the higher distress, the higher decrease in the RF, but no significant impact of the RF on distress). For brooding, I found that negative homeostasis was the case: Lower brooding before exams was associated with a lower increase in mental distress during exams, while higher mental distress before exams was associated with a higher chance of more abstract, negative-focussed and repetitive thinking during exams. However, I only found a mutualistic effect for the time from before to during exams, not for the time after the exams. For self-esteem non-mutualistic strength-dominance was the case: There was no mutualistic effect with mental distress, rather, self-esteem seemed to mitigate increase in distress during exams and to foster decrease in distress after exams. Hence, based on those findings it seems that negative homeostasis or strength-dominance are likely scenarios. Neither negative homeostasis nor strength-dominance does however align with the risk homeostasis theory, in fact, those findings rather suggest that leveraging RFs may not be a flawed idea.

A limitation based on my work is that I used a natural stressor which is likely to impact both the RFs and distress. A scenario that may be more specific to testing the risk homeostasis theory for the relationship between RFs and mental distress would be to directly "manipulate" or "intervene" on either distress or the RFs. In this case, one would need to directly target and either increase distress symptoms, decrease distress symptoms (e.g. symptom-focused psychotherapy), increase the level of RFs (e.g. strength-based interventions), or decrease the level of RFs. However, increasing distress and reducing RFs may well be unethical. I believe that it would be highly interesting to test increasing RFs, such as via a strength-based intervention, as this would align most closely with the mental health resilience framework. The four possible outcomes discussed in the previous paragraph are depicted in Figure 8.2 for the option of decreasing distress symptoms (left panel) and for increasing RFs (right panel). Of note, for simplicity I depict theoretically possible path-models (instead of more complex change score models as discussed in Chapter 7).

It would seem plausible that negative homeostasis (i.e. the higher the RF, the lower distress) would hold, hence that increasing RFs would have a positive effect on mental health. This would then provide solid evidence against the risk homeostasis idea and indicate that leveraging RFs is not a flawed idea. Positive homeostasis (i.e. the higher the RF, the higher distress) would probably seem the least likely of the four outcome options, as this would largely differ from my findings in Chapter 7. It would also not align with the findings of the other chapters which clearly show that RFs and distress are concurrently as well as prospectively negatively associated with each other. Hence, based on my research I would not suspect that the risk homeostasis theory would hold (or at least not in the way I have translated the verbal theory into a model). Whichever the outcome would be, a research design as depicted in Figure 8.2 would likely provide a clearer answer to Bonnano and colleagues' (2011; p. 529) question "Can we, in fact, make people more resilient, or is resilience building a flawed idea?". To fully answer the question one may additionally need to test the model in a group of individuals with and in a group of individuals without adversity, to see whether the effect of strength- or resilience-building depends on exposure to adversity.

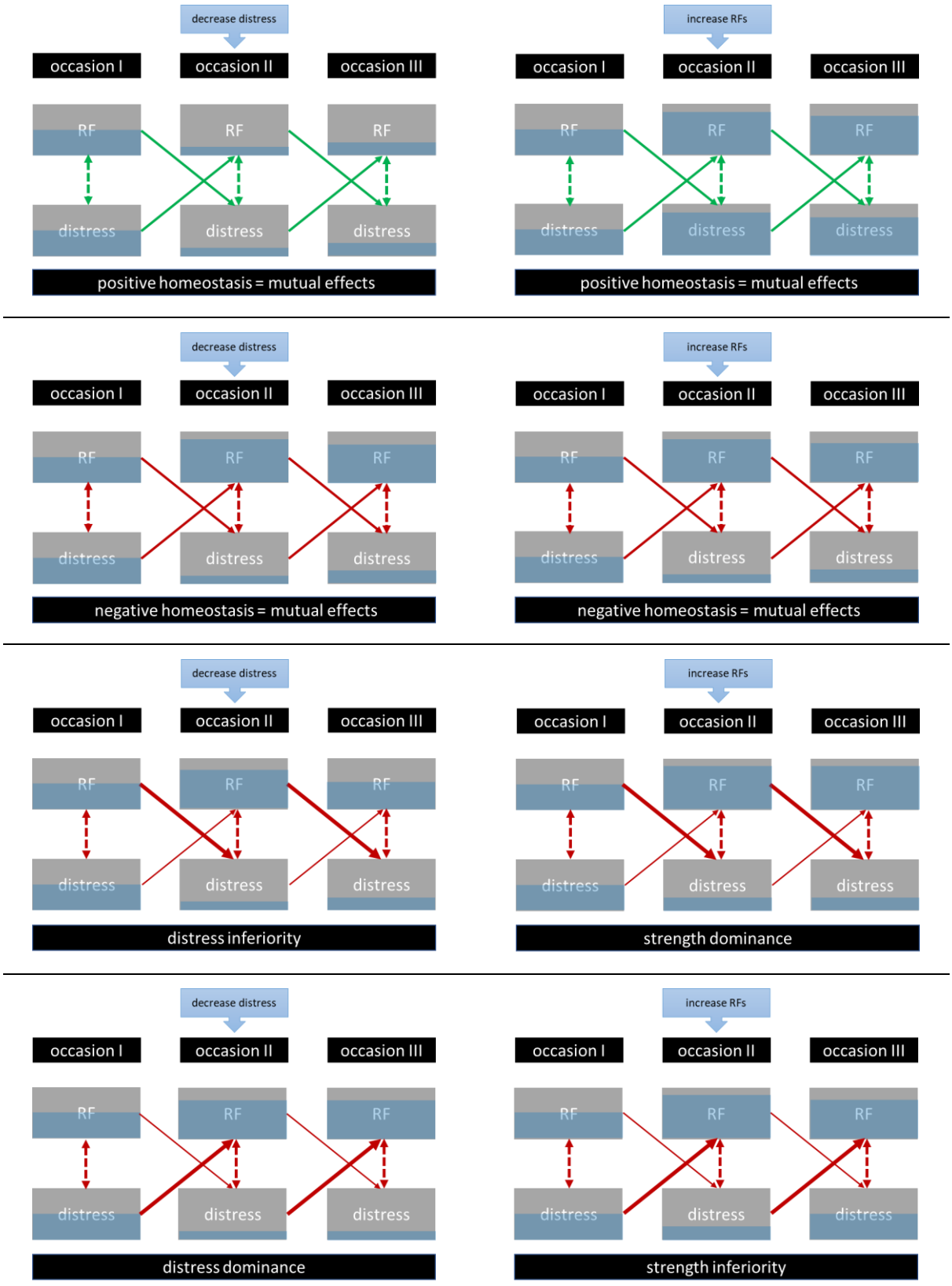



*Figure 8.2.* This Figure depicts different path-model scenarios of a longitudinal intervention study with three time points. The left panel depicts an intervention that at the second time point intervenes on the (mental) distress symptoms. The right panel depicts an intervention that at the second time point intervenes on the resilience factor (RF). The two upper panels depict a homeostasis scenario in which the RF and mental distress impact each other to a similar extent. The third panel depicts a strength dominance (or distress inferiority) scenario, in which the RF

exerts more effect on distress than distress on the RF. The lower panel depicts a distress dominance (or strength inferiority) scenario, in which mental distress exerts more effect on the RF than the RF on distress. The arrows indicate in which direction the effect operates. Green arrows = positive relationship, red arrows = negative relationship. Solid arrows = longitudinal relationships, dashed arrows = cross-sectional relationships. Of note, I do not depict autoregressive relationships to simplify the speculative hypotheses, yet, they are necessary for the eventual evaluation. Blue shadow with which the respective RF or distress boxes are filled = activation level. Please note, this Figure is just a simplified depiction to sketch potential scenarios, other scenarios are possible.

One could even take such a research endeavour a step further and not only test the risk homeostasis theory for mental health with data models, but one could also develop a formalized risk homeostasis model for mental health (i.e. a computational model of risk homeostasis for mental health; Cramer et al., 2016). More specifically, one would have to define and calculate a formal (implied data) model with simulated data based on differential equations (or another formal framework, e.g. agent-based modeling) and compare this against the empirical data model (Haslbeck, Ryan, Robinaugh, Waldorp, & Borsboom, 2019). The implied data model, although not based on empirical data, is relevant as it enables the simulation of the interaction between mental health and RFs that would be expected given the formal risk homeostasis theory for mental health (Burger et al., 2020; Haslbeck et al., 2019). Together such knowledge (i.e. the discrepancy between both models) can be used to update and improve the formal theory, which eventually can enrich our understanding of the interaction between mental health symptoms and RFs, and thus enhances prediction and control of those interactions (Haslbeck et al., 2019).

## 8.7 HOW MY RESEARCH CAN INFORM TRANSLATIONAL RESEARCH ON MENTAL HEALTH PROMOTION

I believe that there are at least three ways in which my findings can be applied to mental health promotion, namely by informing: (a) risk detection and mental health screening, (b) psychoeducation, and (c) translational research on psychological interventions.

### 8.7.1 How Can RFs Inform Risk Detection and Mental Health Screening?

The first way in which knowledge on RFs can be leveraged is for risk detection and mental health screening. In contrast to very young children and very old adults, adolescents and young adults have often less frequent health checks, and if they are generally physically healthy, see general practitioners (GPs) less often (Patel et al., 2007). Therefore, particularly for adolescents and young adults, mental health screening may need to happen on a level other than at the GP. A potentially better place may be schools, universities or the work-place. However, those places would likely come with a stigma risk. Another option would be to simply screen people online, “at home” (provided internet access is available). At all of these places, one could use (online) self-esteem and brooding instruments in a regular fashion to efficiently check the potential risk levels for mental health problems. For example, previous research by Young and Dietrich (2014) employed the same brooding subscale as used in my research (5 items of the RRS; Treynor et al., 2003) and detected a screening accuracy of 91 percent

for concurrent depressive symptoms in young adolescents. My research has shown that RFs can identify for only two in three adolescence whether they will have low, moderate or high distress three years later. Yet, firstly, distress did not predict itself better over the course of three years. Secondly, I studied the vulnerable period between the ages of 14 and 17, during which as much as fifty percent of all mental health problems start emerging (World Health Organization, 2018). Hence, as RFs seem to be similarly predictive for subsequent mental distress, as mental distress itself, RFs may potentially be promising as crude mental-health screens.

That said, much more research is needed to show whether the advantages of using RF screens outweigh the disadvantages. The National Screening Board of Public Health England (Public Health England, 2015) put forward criteria which need to be fulfilled by screening tools to be deemed as viable (see Box 3). I shall briefly outline which criteria RF screens would likely tick off and which criteria may well be more difficult to be realized – a complete debate of this topic is however beyond the scope of this discussion.

**BOX 3: CRITERIA FOR DEVELOPING VIABLE SCREENING TOOLS, AS PUT FORWARD BY PUBLIC HEALTH ENGLAND (2015)**

1. the to-be-predicted disease needs to be screening-worthy
2. the disease characteristics and trajectories should be adequately known
3. the screen needs to be "simple, safe, precise and validated" (Public Health England, 2015, p. 1)
4. the population-based descriptive statistics should be established, and classification thresholds should be evaluated and agreed
5. the screen needs to be adequately acceptable (i.e. on the social, clinical and ethical level) for the tested population
6. the programme in which the screening procedure is embedded in needs to be adequately acceptable (i.e. on the social, clinical and ethical level) for the screening staff and the target group
7. there needs to be a diagnostic follow-up test
8. there needs to be an intervention option that can decrease the disease morbidity (as confirmed by randomized controlled trials)
9. screening benefits need to offset negative physical and mental consequences
10. screening should be cost-effective

The first two criteria are that the to-be-predicted disease needs to be screening-worthy, and the disease characteristics and trajectories should be adequately known (Public Health England, 2015). Although more knowledge on the aetiology of transdiagnostic mental distress and internalizing disorders would clearly be an advantage, there is some solid knowledge regarding the aetiology, possible clinical trajectories and the health and well-being burden (see section 8.5 and 8.6). Moreover, Newlove-Delgado and Ford (2020) discuss that young people start experiencing sub-threshold symptom levels on average two years before meeting diagnostic criteria, which underpins the importance of early and repeated screening. The third and fourth criterion state that the screen needs to be "simple, safe, precise and validated" (Public Health England, 2015, p. 1) and that classification

thresholds should be evaluated and agreed. Of course, the prediction accuracy of RF screens would need to be substantially improved and replicated. Moreover, clinical thresholds would need to be defined, tested and agreed. Criterion five and six require that the screening tool is adequately acceptable (i.e. on the social, clinical and ethical level) for the tested population as well as for the screening staff (Public Health England, 2015). RF screens may well be acceptable for the population as they are expected to have a relatively low stigma-risk and may for some people be more acceptable than symptom-focussed screens. However, those are so far tentative conjectures which need to be confirmed. Equally, it would need to be tested how such screens could best be realized on a large scale. As discussed above, one potential option, particularly for adolescents, could be to establish school-based screening programs. Similarly, university-based screening or work-place screening in at-risk workplaces (e.g. military, fire brigade or medical staff) would seem realistic as well. Particularly as screening could be conducted online, the burden on screening staff would seem minimal.

The seventh criterion, that a diagnostic follow-up test needs to be in place (Public Health England, 2015), is provided by the DSM 5 guidelines and related diagnostic interviews (American Psychiatric Association, 2013; First, Williams, Karg, & Spitzer, 2016). The eighth criterion entails that there needs to be an intervention option that can decrease the disease morbidity (Public Health England, 2015). Although there are several intervention options for the prevention and treatment of (internalizing) mental health disorders (also via RFs; see section 8.7.3 for details), access to those interventions is limited. Therefore, the critical question remains whether mental health screening could at all be beneficial if mental health interventions cannot be offered on a larger scale (Gilbody, Sheldon, & Wessely, 2006). Critics may also rightfully argue that screening increases the identification of people in need for intervention, which may make intervention access even more difficult for people with the highest need (Anderson et al., 2018; Gilbody et al., 2006). Criterion nine requires that screening benefits need to offset negative physical and mental consequences (Public Health England, 2015). So far there is very little research, but to the best of my knowledge research has not indicated that the assessment of RFs causes physical or mental harm. That said, there is always a risk of false-negative screening results, in which case people are at-risk but are not identified as such. This may of course lead to a false sense of reassurance (Newlove-Delgado & Ford, 2020). Equally, there is the risk of false-positive screening results, in which case people are identified as being at-risk, but are not actually at risk. In this case, people are likely to experience an unnecessary emotional burden (Newlove-Delgado & Ford, 2020). Both false-positives and false-negatives are always a risk of screening procedures and can only be limited by the development of precise, reliable and valid psychometric screens (Newlove-Delgado & Ford, 2020). Thus, future research would be critically needed to investigate how we can improve the screening accuracy. The tenth criterion entails that screening should be cost-effective (Public Health England, 2015). A recent systematic review suggested that mental health screening costs may not outweigh the costs for treatment as usual in young people, but that there currently is too little knowledge to draw a valid conclusion (Anderson et al., 2018). Moreover, this knowledge synthesis was mainly based on disorder-focussed screens and not on RF screens.

Taken together, there is a chance that RFs may be viable as mental health screens – but, besides the remaining question whether RF screens are indeed less prone to stigma risks and better

acceptable than symptom-focussed screens, screening benefits may only outweigh the costs if (a) we succeed in increasing and replicating the screening accuracy, (b) young people who screen positive but are below-threshold cases for mental disorders can access evidence-based prevention programs, and if (c) young people who screen positive and indeed qualify for a diagnosable mental disorder can access evidence-based treatment programs.

### 8.7.2 How Can RFs Inform Psychoeducation?

A second way in which knowledge on RFs may inform mental health promotion is psychoeducation. For example, social workers, teachers, pastoral support, counsellors, and psychotherapists could use such knowledge to not only inform about symptoms (i.e. their potential origin, interpretation and treatment), but also about skills and resources that promote good mental health. Psychoeducation can vary in level of depth and can for example be delivered in form of flyers, brochures, education websites, audio-visual aids, feedback on test results, educative phone calls or emails, and information sessions (Donker, Griffiths, Cuijpers, & Christensen, 2009). Hence, psychoeducation can be cost- and time-efficiently delivered to a large audience – such as in schools, universities, at the work-place, or in community centres – and can therefore reach wide and far (Donker et al., 2009). Particularly online material seems advantageous when fast access is essential (Donker et al., 2009). Although it is very likely that day-to-day psychoeducation often already includes information on RFs, empirical support for the actual usefulness of RFs should further underpin and potentially increase the inclusion of such information in psychoeducation.

More specifically, insights into RFs can firstly be used to inform about the intra-personal promotive skills that can help to prevent mental health problems (e.g. low brooding and high self-esteem) and can secondly inform about the importance of a positive and supportive environment (e.g. friendship and family support, and family cohesion). For example, adolescents who grow up with a parent suffering from severe mental health problems may be advised to also seek support from other family members, extended family, or friends, to ensure that there is sufficient background support when times at home are tough. Similarly, parents could be informed that not only intra-personal skills influence the mental health of their kids, but that family support and cohesion are also relevant factors that go together with mental health. It is of course equally important to explain that the family has a limited influence and is not necessarily able to resolve poor mental health. Donker and colleagues (2009) showed that a basic level of psychoeducation can already have a relevant effect ($d = 0.20$) in the reduction of depression and distress symptomatology. Accordingly, it may well be possible that supplementing psychoeducation with knowledge on RFs is fruitful too. That said, the findings discussed in this thesis that can theoretically inform psychoeducation need ideally first to be replicated to ensure reliability and external validity.

### 8.7.3 How Can RFs Inform Psychological Interventions?

When evaluating how RFs can inform psychological interventions, several considerations are necessary, such as for which interventions RFs could be most fruitful, for whom RFs would be

advantageous, at which point in time targeting RFs may be most efficient, and in which settings interventions that target RFs can be offered and leveraged.

The *first* consideration "*for which interventions RFs could be most fruitful*" is important as this enables us to identify how RFs can best be embedded into already existing intervention modules and/or manuals. As outlined in the introduction, i.e. Chapter 1, knowledge on RFs may be particularly interesting for mental health interventions that focus on enhancing positive cognitions, emotions, and behaviours rather than on decreasing symptoms; so-called positive psychology interventions (Bolier et al., 2013). A notable strength of positive psychology interventions is that they focus on aspects that are applicable when symptoms have not yet evolved (i.e. prevention), can fill in those areas that are not touched by classical symptom-based interventions once symptoms have evolved (i.e. treatment), and can sustain and enhance mental health after successful symptom interventions (i.e. relapse prevention). In the introduction I discuss several positive psychology interventions of which two seem particularly suited for the incorporation of RFs. One is Seligman's Positive Psychotherapy (PPT) intervention which generally focusses on fostering positive feelings, intra-personal skills and resources, as well as a meaningful life (Seligman et al., 2006). PPT operates with a specific list of strengths from which core strength can be identified. Importantly, many of those strengths seem to be personality characteristics (e.g. creativity, curiosity, bravery, social intelligence, fairness, prudence, humour, and spirituality) rather than amenable skills and resources (Seligman et al., 2006). Hence, those strengths seem particularly profitable when they are pre-existent and can be capitalized on. Yet, for individuals for whom it is difficult to recruit or exploit those characteristics, it may well be fruitful to aim for the enhancement of more amenable skills, such as improving self-esteem as well as identifying and reducing brooding, or switching the ruminative thoughts from negative to positive.

The other positive psychology intervention is Fava's Well-Being Therapy (WBT) which focusses on fostering or reinstating well-being, aiming for a full recovery and reducing the risk of relapse, instead of reducing and overcoming symptoms (Fava & Tomba, 2009). As laid out by Fava and Tomba (2009; p. 1910) one aim of WBT is "*that criteria can be established to distinguish valid and helpful positive thinking from unrealistic expectations and that promotion of psychological well-being may result in decrease of distress and higher levels of resilience to environmental circumstances*". WBT has an intervention phase in which it aims to fosters (a) environmental mastery, (b) a purpose in life, (c) autonomy, (d) self-acceptance, (e) personal growth, and (f) positive relationships with others (Fava & Tomba, 2009). My findings can inform WBT, for example because they show that both friendship support and family support are significantly related with concurrent and future mental health problems, providing empirical evidence for the positive effect of positive relationships. Moreover, my findings showed that distress tolerance, which is part of environmental mastery, is also related to concurrent and future mental health problems. However, when I used multiple RFs simultaneously as predictors for mental health, self-esteem and brooding RFs had the strongest effects. Hence, those two RFs may potentially be most fruitful and seem conceptually to link to many of the skills WBT aims for. For example, brooding is defined as "negative self-evaluative thinking (e.g., 'Why can't I handle things better?') and comparative thinking about the self (e.g. 'Why do I have problems other people don't have?')" (p. 2 in Watkins & Roberts, 2020), and can thus be a form of moody pondering about autonomy

and environmental mastery. Similarly, self-esteem concerns questions such as “Do you care about being a . . . good romantic partner, a good parent, and a good friend? Do you desire to make valuable contributions to your profession? Do you want to behave morally and competently in the domains of your life?” (p. 48 in Greenberg, 2008), and is thus closely related to positive relationships with others, a purpose in life, personal growth and/ or self-acceptance. As my network analyses revealed that self-esteem and brooding are clearly linked with social support and distress tolerance, it may even be possible that targeting self-esteem and brooding has a positive effect on other well-being skills. Yet, this is of course highly speculative and needs to be formally tested in translational research.

Besides the common positive-psychology interventions, there is some evidence that shows that building an intervention carefully around a specific RF may also be lucrative. For example, there is preliminary but good evidence for rumination-focussed CBT, particularly for people at-risk for internalizing disorders (Cook et al., 2019; Watkins, 2015). Similarly, self-esteem has also been found to be a successful intervention theme for preventing or mitigating internalizing disorders in young people (Bos et al., 2006). Moreover, one could argue that common CBT techniques resemble or lead to enhancing specific RFs. For example, exposure techniques may well result in building distress tolerance; or social skills training may well result in enhancing skills for seeking and receiving friendship and family support. Furthermore, RFs may inform symptom-focussed transdiagnostic interventions, which are not set out to treat a specific disorder (e.g. major depressive disorder), but can be leveraged for various disorders or a disorder category (e.g. internalizing disorders). The question then is, for which type of transdiagnostic treatments RFs may lend themselves best. Dalgleish and colleagues (2020) cluster transdiagnostic treatments into modular intervention and universal intervention classes, of which the former one contains a selection of treatment modules that can be individually chosen and compiled for the patient, whereas the latter focuses on a multipurpose treatment protocol applicable to a wide range of diagnoses. Technically, RFs could inform both transdiagnostic intervention classes, as a positive-psychotherapy module could be fit into both an intervention module as well as into a universal protocol. Yet, as it has been suggested that a strength-focus is more likely to be effective before the full manifestation of the symptoms (prevention) or when the major symptoms have already been targeted (at the end of symptom-treatment or in relapse prevention), a positive-psychotherapy module may lend itself better for modular interventions, so that it simply could be skipped for patients for whom it would not be useful or at least not a priority.

The *second* consideration “*for whom RFs would be advantageous*” seems crucial as it helps to pinpoint the frame and relevance of including RFs in psychological interventions. My findings provide some preliminary conjectures. Generally, my findings seem to suggest that most RFs do not significantly differ in their effect of mitigating distress between groups of adolescents with and without CA. Yet, friendship support, family cohesion, and distress tolerance seemed to attenuate subsequent mental health problems only significantly in individuals with, but not in individuals without exposure to CA (when controlling for the other RFs). Importantly however, also for those three RFs there was no significant and at best a marginal difference between individuals with and without CA. Thus, further research is needed before we know whether those three RFs are or are not particularly fruitful for individuals with a CA history. That said, I found rather robust evidence for low brooding being effective in mitigating

mental distress in individuals with as well as in individuals without exposure to CA. Hence, particularly brooding may be fruitful in situations when it cannot (ethically or practically) be assessed which individuals have a history of CA, such as sometimes in school-based group interventions. However, it is of utmost importance to test whether my findings hold in replication and whether the identified RFs are actually useful when targeted in interventions.

The *third* consideration is "*the time point at which targeting RFs may be most efficient*". As indicated above, interventions that target RFs seem to be particularly helpful in prevention for individuals at risk, before mental health problems have significant impact, and for relapse prevention, when the aim is to move on from symptom-based treatment. For example Fava and Tomba (2009; p. 1916) report that "it is quite difficult to apply WBT in an acutely ill patient (e.g., in a major depressive episode) because the amount of negative thoughts may be at that stage overwhelming. WBT appears to be more suitable for addressing psychological issues that other therapies have left unexplored". Translational research has provided preliminary evidence that PPT and WBT seem to be effective both as preventative intervention and as relapse prevention intervention (Bolier et al., 2013; Fava & Tomba, 2009). Moreover, rumination focused cognitive behaviour therapy has been shown to be a promising prevention intervention for adolescents at risk for internalizing mental health problems (Watkins, 2015). For self-esteem there seems to be some, as of yet, preliminary evidence for its suitability as prevention target (Bos et al., 2006).

Given that we have seen how the here acquired knowledge on RFs can theoretically inform translational intervention research, the fourth and last consideration is "*in which settings interventions that target RFs can be offered and leveraged*". Strength-based intervention approaches appear to be less stigma prone than symptom-based intervention approaches and may therefore be particularly lucrative for school, university, community, or even work-place settings. Those approaches may potentially also attract new target audiences who would refuse symptom-based interventions. Particularly strength-based approaches that can be delivered as group or online-based interventions may be both easily accessible and cost-effective (Bolier et al., 2013; Seligman et al., 2006). All in all, further evaluation of targeting RFs in (relapse) prevention seems relevant and promising.

## 8.8 CONCLUDING REMARKS

In sum, the here presented doctoral research was aimed at providing and extending empirical knowledge on intra- and inter-personal skills and resources that promote mental health in young people with and without a history of adversity. Before concluding my doctoral thesis I would like to repeat what I stated in the introduction: I have aimed to the best of my ability to shape an as accurate, robust, and reliable picture of the findings as possible, but the "answers suggested here are examples, not conclusions; some will turn out to be wrong" (see p. xii in Nesse, 2019). That said, I believe that my doctoral aim was successfully met and that my research has shed promising light on the complex picture of psychosocial RFs, which can be leveraged to inform mental health screening, psychoeducation, prevention, treatment, as well as transdiagnostic mental health and resilience theory. Specifically, my

research has revealed (a) that a holistic framework, capturing multiple ecological levels, is likely to have the most effective impact on mental health, (b) that RFs can be described as promotive system, which seems to be disadvantaged in adolescents with compared to adolescents without a history of adversity (particularly proximally after the adversity exposure), (c) that in adolescents with a history of adversity protective pathways between RFs and mental distress seem not only to be disadvantaged, but the disadvantageous level seems to be carried forward from earlier to later adolescence, which may help explain why exposure to adversity is frequently found to have a lasting effect on mental health, (d) that brooding and self-esteem seem to be particularly promising indicators for transdiagnostic mental health problems, and their value as risk or mental-health screens should be further evaluated, (e) that it is helpful and at times perhaps even crucial to take precipitating factors, such as an adversity history, into account, when the aim is to identify the most promising set of promotive factors, and (f) that brooding and self-esteem RFs may well be suitable transdiagnostic intervention targets which should be tested in translational research.

Although the acquired knowledge on RFs has provided various answers, it may have shed light on even more questions than answers. Future research could for example be aimed at (a) developing a better and more holistic childhood adversity measure, (b) performing more research on well-being in addition to mental health, (c) tracking RFs over the life course, (d) investigating cultural and environmental RFs, (e) studying RFs on an idiographic level, (f) examining the value of RFs for risk-screening and psychoeducation, and (g) testing whether intervening on RFs is a flawed or a useful idea (e.g. via testing strength-based interventions). Therefore, I believe that from here onwards we should passionately look into a bright future full of inspiring research questions on the complex nature of strength-focussed mental health promotion, whether with a magnifying glass or from a bird’s eye view.

I like to thank all readers for their interest and for sticking with me. I hope that I could inspire you to embrace already available skills and resources, or to acquire new ones, to seek out for support and to support others. Take care, and dare to struggle and to flourish.

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# APPENDICES

# APPENDIX A
# Appendix Chapter 2

## APPENDIX A.1: CONTENT NOTES

### A.1.1

The inter-rater reliability (IRR) calculation was based on nominal agreement ratings (1 = correct, 0 = incorrect), and was conducted in R with the 'epiR' package (Stevenson, 2016). Given that the data indicated prevalence problems (i.e. marginal distribution of the agreement ratings were substantially more often 'correct' than 'incorrect'; Hallgren, 2012), I utilized Byrt's prevalence-adjusted kappa (Byrt, Bishop, & Carlin, 1993). I calculated Byrt's kappa separately for every abstracted article and averaged the revealed kappas.

### A.1.2

I report the gender ratio for the baseline assessment or the investigated sample. If such information was not available I report information for the first reported assessment time. I report the average, not the absolute, study length and in cases of uncertainty I report the minimal length of the studies. I report the sample sizes which have been used for the analyses. I report the mean ages for the CA assessments, dependent on availability either for the full or for the investigated sample. CA assessment mean ages were provided for 12 of the 22 studies.

### A.1.3

Notably, ego over-control mediated the association between early child maltreatment and alcohol use negatively (Oshri, Rogosch, & Cicchetti, 2013). A lower level of early child maltreatment was associated with less ego over-control and a low level of ego over-control was in turn associated with more alcohol use (Oshri et al., 2013). Given the negative mediation, we did not consider this effect as RF.

### A.1.4

Notably, adolescent-father communication negatively mediated the association between paternal alcohol abuse problems and violation of rules, in girls (Finan, Schulz, Gordon, & McCauley Ohannessian, 2015). A lower level of CA was associated with more adolescent-father communication and more adolescent-father communication was in turn associated with more violation of rules (Finan et al., 2015). Given the negative mediation effect, we did not consider this effect as RF.

## APPENDIX A.2: DATA EXTRACTION ITEM CONTENT TEMPLATE

The data extraction form was partially underpinned by the STROBE report recommendations (Vandenbroucke et al., 2007). The data extraction form covers the following topics: (a) Type of cohort design, (b) length of follow-up period (including time intervals), (c) definition of CA (theoretical and statistical scale used), (d) sample size (if possible per group and assessment occasion), (e) gender, (f) age, (g) demographics (i.e. socio-economic status & ethnicity), (h) measurement type (e.g. questionnaire/interview/objective measure), assessment instrument, and assessment time point for CA, (i) measurement type, assessment instrument, and assessment time point for RF(s), (j) measurements type, assessment instrument, and assessment time point for psychopathology (PP), (k) type (i.e. design/statistics/other) and definition for controlled confounders, (l) used statistics and statistical outcome (i.e. moderation/mediation, analysis method, analysis coefficient, analysis statistic, significance value, confidence interval/standard error, effect size), and (m) conclusion (significance and if appropriate directionality of the effect; see Table A.1).

Table A.1
*Data extraction item content template*

| Item | Item content |
|---|---|
| **1** | Reference |
| **2** | ID |
| **3** | Note |
| **4** | Design |
| **5** | Note Design |
| **6** | Amount of assessment waves |
| **7** | Either indicate the time point for each assessment wave (e.g. T1 January 2010, T2 March 2013; or: T1 age 14, T2 age 17) or the incubation time between the assessments (e.g. T1: baseline, T2: T1 + 3 years, T3: T1 + 5 years) |
| **8** | Gender |
| **9** | CA definition |
| **10** | CA measurement |
| **11** | If CA dichotomous: define control group |
| **12** | Sample size (if possible per occasion; e.g. T1 = 600, T2 = 490) |
| **13** | If CA dichotomous: Sample size per group (and if possible per occasion; e.g. CA+: T1 = 320, T2 = 170; CA-: T1 = 500, T2 = 400) |
| **14** | Mean age (if possible per occasion; e.g. T1 = 14.5, T2 = 17.1) |
| **15** | Note age |
| **16** | If CA dichotomous: Mean age per group (and if possible per occasion; e.g. CA+: T1 = 14.2, T2 = 17.0; CA-: T1 = 14.8, T2 = 17.6) |
| **17** | Demographics: indicate time point (e.g. baseline) |
| **18** | Gender ratio |
| **19** | If CA dichotomous: Gender ratio per group (e.g. CA+: 64% female; CA-: 53% female) |
| **20** | SES |
| **21** | If CA dichotomous: SES per group (e.g. CA+: low; CA-: moderate) |
| **22** | Ethnicity |
| **23** | If CA dichotomous: Main Ethnicity per group (e.g. CA+: 80% white, 20% mixed; CA-: 90% white, 10% mixed) |
| **24** | CA assessment method |
| **25** | Name of CA assessment instrument (e.g. Childhood Trauma Questionnaire) |
| **26** | CA assessed at wave(s): |
| **27** | Amount of RFs |
| **28** | RF 1 assessment method |
| **29** | Name of RF 1 |
| **30** | Name of RF 1 assessment instrument (e.g. Rosenberg Self-Esteem Scale) |
| **31** | RF 1 assessed at wave(s): |
| **32** | RF 2 assessment method |
| **33** | Name of RF 2 |
| **34** | Name of RF 2 assessment instrument (e.g. Rosenberg Self-Esteem Scale) |
| **35** | RF 2 assessed at wave(s): |
| **36** | RF 3 assessment method |
| **37** | Name of RF 3 |
| **38** | Name of RF 3 assessment instrument (e.g. Rosenberg Self-Esteem Scale) |
| **39** | RF 3 assessed at wave(s): |

| | |
|---|---|
| **40** | Amount of PP measures |
| **41** | PP assessment method 1 |
| **42** | Name of PP 1 |
| **43** | Name of PP assessment instrument 1 (e.g. Beck's Depression Inventory) |
| **44** | PP 1 assessed at wave(s): |
| **45** | PP assessment method 2 |
| **46** | Name of PP 2 |
| **47** | Name of PP assessment instrument 2 (e.g. Beck's Depression Inventory) |
| **48** | PP 2 assessed at wave(s): |
| **49** | PP assessment method 3 |
| **50** | Name of PP 3 |
| **51** | Name of PP assessment instrument 3 (e.g. Beck's Depression Inventory) |
| **52** | PP 3 assessed at wave(s): |
| **53** | Confounders measured and controlled for by: Design, statistics, or other (specify in description) |
| **54** | Specify confounders (e.g. SES, age, gender, etc.) |
| **55** | Specify PP: |
| **56** | Specify RF: |
| **57** | Choose: Mediation or Moderation |
| **58** | Analysis method e.g. regression, SEM, GEE, multilevel model + (MODERATION: (if neccesarry amount of RF main effects), amount of moderator(s), amount of RF moderator(s); MEDIATORS: amount of mediator(s), amount of RF mediator(s)) |
| **59** | Analysis coefficient (e.g. beta) |
| **60** | Analysis statistic (e.g. t or z) |
| **61** | Significance (e.g. p value) |
| **62** | CI or SE |
| **63** | Effect Size |
| **64** | Note |
| **65** | Specify PP: |
| **66** | Specify RF: |
| **67** | Choose: Mediation or Moderation |
| **68** | Analysis method e.g. regression, SEM, GEE, multilevel model + (MODERATION: (if neccesarry amount of RF main effects), amount of moderator(s), amount of RF moderator(s); MEDIATORS: amount of mediator(s), amount of RF mediator(s)) |
| **69** | Analysis coefficient (e.g. beta) |
| **70** | Analysis statistic (e.g. t or z) |
| **71** | Significance (e.g. p value) |
| **72** | CI or SE |
| **73** | Effect Size |
| **74** | Note |
| **75** | Specify PP: |
| **76** | Specify RF: |
| **77** | Choose: Mediation or Moderation |
| **78** | Analysis method e.g. regression, SEM, GEE, multilevel model + (MODERATION: (if neccesarry amount of RF main effects), amount of moderator(s), amount of RF moderator(s); MEDIATORS: amount of mediator(s), amount of RF mediator(s)) |
| **79** | Analysis coefficient (e.g. beta) |
| **80** | Analysis statistic (e.g. t or z) |
| **81** | Significance (e.g. p value) |
| **82** | CI or SE |
| **83** | Effect Size |
| **84** | Note |
| **85** | Specify PP: |
| **86** | Specify RF: |
| **87** | Choose: Mediation or Moderation |
| **88** | Analysis method e.g. regression, SEM, GEE, multilevel model + (MODERATION: (if neccesarry amount of RF main effects), amount of moderator(s), amount of RF moderator(s); MEDIATORS: amount of mediator(s), amount of RF mediator(s)) |
| **89** | Analysis coefficient (e.g. beta) |
| **90** | Analysis statistic (e.g. t or z) |
| **91** | Significance (e.g. p value) |
| **92** | CI or SE |
| **93** | Effect Size |
| **94** | Note |
| **95** | Specify PP: |
| **96** | Specify RF: |
| **97** | Choose: Mediation or Moderation |
| **98** | Analysis method e.g. regression, SEM, GEE, multilevel model + (MODERATION: (if neccesarry amount of RF main effects), amount of moderator(s), amount of RF moderator(s); MEDIATORS: amount of mediator(s), amount of RF mediator(s)) |
| **99** | Analysis coefficient (e.g. beta) |
| **100** | Analysis statistic (e.g. t or z) |
| **101** | Significance (e.g. p value) |
| **102** | CI or SE |
| **103** | Effect Size |

| | |
|---|---|
| **104** | Note |
| **105** | Specify PP: |
| **106** | Specify RF: |
| **107** | Choose: Mediation or Moderation |
| **108** | Analysis method e.g. regression, SEM, GEE, multilevel model + (MODERATION: (if neccesarry amount of RF main effects), amount of moderator(s), amount of RF moderator(s); MEDIATORS: amount of mediator(s), amount of RF mediator(s)) |
| **109** | Analysis coefficient (e.g. beta) |
| **110** | Analysis statistic (e.g. t or z) |
| **111** | Significance (e.g. p value) |
| **112** | CI or SE |
| **113** | Effect Size |
| **114** | Note |
| **115** | Analysis 1: Conclusion |
| **116** | Analysis 2: Conclusion |
| **117** | Analysis 3: Conclusion |
| **118** | Analysis 4: Conclusion |
| **119** | Analysis 5: Conclusion |
| **120** | Analysis 6: Conclusion |
| **121** | Name of RF 1 |
| **122** | To which resilience factor category belongs the RF 1? |
| **123** | To which resilience factor domains belongs RF 1? NB: Indicate **ALL** domains that are applicable: e = emotional, b = behavioural, s = social, c = cognitive. E.g.: e, s. |
| **124** | Name of RF 2 |
| **125** | To which resilience factor category belongs the RF 2? |
| **126** | To which resilience factor domains belongs RF 2? NB: Indicate **ALL** domains that are applicable: e = emotional, b = behavioural, s = social, c = cognitive. E.g.: e, s. |
| **127** | Name of RF 3 |
| **128** | To which resilience factor category belongs the RF 3? |
| **129** | To which resilience factor domains belongs RF 3? NB: Indicate **ALL** domains that are applicable: e = emotional, b = behavioural, s = social, c = cognitive. E.g.: e, s. |

## APPENDIX A.3: QUALITY ASSESSMENT ITEM CONTENT TEMPLATE

Downs and Black's (1998) scale is recommended for the assessment of randomized, as well as non-randomized studies (Deeks et al., 2003). Given that we exclusively evaluated cohort studies we excluded 9 of 27 items, which are specific to randomized studies (i.e. adverse intervention effects, quality of the description of the used intervention, same recruitment for all intervention groups, representativeness of treatment facilities, intervention compliance, intervention blinding, randomization procedure, randomization concealment and power; Downs & Black, 1998). We duplicated one item, which assesses the accuracy of the outcome measure (i.e. psychopathology), twice, to also assess the accuracy of the CA and RF variables. Accordingly, the adapted quality rating scale contained 20 items (see Table A.2).

Table A.2
*Quality assessment item content template: adapted version of Downs and Black's* (1998) *quality rating scale*

| **Item** | **Item content**<br>***NB: content of the original items from Downs and Black's*** (1998) ***scale can be retrieved from (i.e. see appendix): http://jech.bmj.com/content/jech/52/6/377.full.pdf*** |
|---|---|
| **Article** | |
| **1** | Reference |
| **2** | ID |
| **3** | Note |
| **Description (1 = yes, 0 = no)**<br>***NB: Questions which start with a * have another rating system*** | |
| **4** | original item 1 |
| **5** | original item 2 |
| **6** | original item 3 |
| **7** | original item 5* 2 = yes, 1 = partially, 0 = no |
| **8** | original item 6 |
| **9** | original item 7 |
| **10** | original item 9 |
| **11** | original item 10 |
| **Validity (1 = yes, 0 = no, 000 = unable to determine)** | |
| **12** | original item 11 |
| **13** | original item 12 |
| **14** | original item 15 (In our case: This is about whether those measuring psychopathology outcome were blind to the resilience factor(s).) |
| **15** | original item 16 |
| **16** | original item 17 (In our case: Are the analyses adjusted for differences between participants regarding the length of follow-up?) |
| **17** | original item 18 |
| **18** | adaption of the original item 20 (In our case: Was the childhood adversity measure used valid and reliable?) |
| **19** | adaption of the original item 20 (In our case: Were the resilience factor measures used valid and reliable?) |
| **20** | original item 20 (In our case: Were the psychopathology measures used valid and reliable?) |
| **21** | original item 21 (In our case: Were the participants in different CA groups recruited from the same population?) |
| **22** | original item 25 |
| **23** | original item 26 |
| **Total score** | |
| **24** | NB: This is an automatic field please do not enter numbers manually! |

## APPENDIX A.4: ANALYSIS METHOD QUALITY ASSESSMENT ITEM CONTENT TEMPLATE

*Analysis method quality assessment: item content template*

| Item | Item content |
|---|---|
| **Article** | |
| **1** | Reference |
| **2** | ID |
| **Sample Size** | |
| **3** | Sample size |
| **4** | Appropriateness sample size: Moderation (NA = no moderation performed , 0 = inappropriate, 1 = appropriate) |
| **5** | Appropriateness sample size: Mediation (NA = no mediation performed , 0 = inappropriate, 1 = appropriate) |
| **RFs tested** | |
| **6** | How many RFs tested in total? |
| **7** | How many RFs significant in total? |
| **8** | How many RFs significant moderators? |
| **9** | How many RFs significant mediators? |
| **10** | Name of significant RFs |
| **Single Versus Multiple RFs** | |
| **11** | Multiple or single RF models? |
| **12** | Amount of moderators per model (0 if not appropriate; i.e. only RF moderators are counted) |
| **13** | Amount of mediators per model (0 if not appropriate; i.e. only RF mediators are counted) |
| **Quality Moderation Analysis** | |
| **14** | Moderation Rating (NA = no moderation performed, ? = not rateable, 1 = no/visual inspection, 2 = correlational post hoc probing, 3 = regression post hoc probing) |
| **15** | Note: Moderation |
| **Quality Mediation Analysis** | |
| **16** | Mediation Rating (NA = no mediation performed, ? = not rateable, 1 = no/direct effect reduction to non-significance, 2 = Sobel (or comparable formulas), 3 = bootstrap) |
| **17** | Note: Mediation |
| **Multiple Testing** | |
| **18** | Correction for multiple testing? (Not Neccesary = NN, no = 0, yes =1) |
| **19** | Note: Correction for multiple testing? |

## APPENDIX A.5: SAMPLE SIZE GUIDELINES

In order to obtain a sample size guideline for interaction effects I conducted a power analysis. The resilience literature shows that whereas multifaceted clusters of CAs explain up to 30 percent of psychopathology indices (Kessler et al., 2010), RF predictors have small to at the best moderate effect sizes (Kalisch et al., 2017). Moreover, research has indicated that interaction effects in social sciences are generally weak (Aguinis, Beaty, Boik, & Pierce, 2005; Champoux & Peters, 1987; Fairchild & McQuillin, 2010). For example, Champoux and Peters (1987) reviewed 23 studies and found that moderation effects account for approximately 1 to 3 percent of the outcome variable (mean $\Delta R^2 = .03$). Similarly, Aguinis and colleagues (2005) found that the 261 reviewed moderation analyses had an average interaction effect size of .01 ($f^2$). Based on those findings and on the fact that many of the reviewed studies include additional covariates or interaction effects, which requires larger sample sizes, I calculated the sample size for a moderation analysis with a moderate total effect ($f^2 = .15$). The analysis was conducted in R with the package 'pwr' (Champely, 2017). I specified two main effects, one interaction effect (u = 3), a moderate effect size of $f^2 = .15$ (i.e. based on the above described findings and on Cohen's effect size criteria; J. Cohen, 1988), an alpha level of .05, and a power threshold of .80. The analysis showed that a minimum of 77 participants is required (v = 72.71; $N = v + (u+1)$; $N = 72.71 + 4$) to detect a moderate effect.

## APPENDIX A.6: STUDIED TYPES OF CHILDHOOD ADVERSITY AND PSYCHOPATHOLOGY

*Studied types of childhood adversity and psychopathology*

| CHILDHOOD ADVERSITY | | | |
|---|---|---|---|
| **Childhood Maltreatment** | **Intra-Family Adversity** | **Community Adversity** | **Clustered Life Adversities** |
| emotional abuse (Banducci, Lejuez, Dougherty, & MacPherson, 2017; Calvete, 2014; Hankin, 2005; Oshri et al., 2013; Walter, Horsey, Palmieri, & Hobfoll, 2010; You & Lim, 2015) | marital distress/conflict (Cui & Conger, 2008) | ethnic-political conflict (Dubow et al., 2012; Qouta, El-Sarraj, & Punamäki, 2001; Shahar & Henrich, 2015) | adverse life experiences (Boyes, Hasking, & Martin, 2015; Masten et al., 1999) |
| sexual abuse (Dennison et al., 2016; Hankin, 2005; Hébert, Cénat, Blais, Lavoie, & Guerrier, 2016; Oshri et al., 2013; Walter et al., 2010) | parental problem drinking (Finan et al., 2015) | community violence (Hardaway, Sterrett-Hong, Larkby, & Cornelius, 2016) | |
| physical abuse (Dennison et al., 2016; Hankin, 2005; Lansford et al., 2006; Oshri et al., 2013; Walter et al., 2010; You & Lim, 2015) | aggressive parenting behaviour (Gaté et al., 2013) | | |
| emotional neglect (Oshri et al., 2013; You & Lim, 2015) | parental violence (Jester, Steinberg, Heitzeg, & Zucker, 2015) | | |
| physical neglect (Oshri et al., 2013; You & Lim, 2015) | parental mental health problems (Klasen et al., 2015) | | |
| | stressful family-level life events (Hicks et al., 2014) | | |
| | accumulated family adversity (van Harmelen et al., 2016) | | |

| PSYCHOPATHOLOGY | |
|---|---|
| **Disorder Types** | **Clustered Types of Psychopathology** |
| anxiety symptoms (Banducci et al., 2017; Hankin, 2005; Shahar & Henrich, 2015)/ social anxiety symptoms (Calvete, 2014) | psychological distress (Boyes et al., 2015; Hébert et al., 2016) |
| depressive symptoms (Calvete, 2014; Dennison et al., 2016; Gaté et al., 2013; Hankin, 2005; Klasen et al., 2015; Shahar & Henrich, 2015; van Harmelen et al., 2016) | mental well-being (Cui & Conger, 2008; Hébert et al., 2016) |
| posttraumatic stress symptoms (Dubow et al., 2012; Qouta et al., 2001; Walter et al., 2010) | Externalizing (Cui & Conger, 2008; Finan et al., 2015; Hardaway et al., 2016; Lansford et al., 2006; Oshri et al., 2013; Qouta et al., 2001) |
| substance (ab)use symptoms (Hicks et al., 2014; Jester et al., 2015; Oshri et al., 2013) | Internalizing (Cui & Conger, 2008; Hardaway et al., 2016; Lansford et al., 2006; Oshri et al., 2013; Qouta et al., 2001) |
| conduct symptoms (Masten et al., 1999; Shahar & Henrich, 2015; You & Lim, 2015) | |

# APPENDIX B
# Appendix Chapter 3

## APPENDIX B.1: METHODOLOGICAL RATIONALE

The network models we estimated for the current research are based on (a) correlations between the RFs (see Appendix B.4) and (b) regularized partial correlations between RFs (see Figure 1 in the main text). The main text focuses on the discussion of regularized partial correlation networks for two reasons. First, partial correlations between RFs indicate to which extent two RFs are associated with each other, while controlling for all other RFs in the network. This way, we get an indication about which RFs predict each other and to which extent (Epskamp, Waldorp, Mõttus, & Borsboom, 2018). Second, we applied regularization to the partial correlations, as no partial correlation between RFs will be exactly zero (Epskamp, Waldorp, et al., 2018). Thus, regularization was applied to set very small partial correlations, which are likely to be false positives, to exactly zero (i.e. those interrelations are not depicted in the networks), resulting in potentially sparse models that exclusively depict the meaningful RF interrelations (Epskamp, Waldorp, et al., 2018).

To obtain the partial correlations for the discussed networks, it is necessary to (1) estimate the variance-covariance matrix, (2) take the inverse of this matrix (called precision matrix), and (3) standardize the precision matrix (Epskamp, Waldorp, et al., 2018). The standardized precision matrix then contains the partial correlations between the RFs, corrected for the respective other RFs in the matrix. In statistical terms, the corresponding graphical model for normally distributed, continuous variables is the Gaussian Graphical Model (GGM). Epskamp, Waldorp and colleagues (2018) have shown that the GGM network model is closely related to least-squares regression. Similarly, GGM and structural equation models (SEM) are quite comparable as they both entail a constrained covariance structure (Epskamp, Rhemtulla, & Borsboom, 2017). Moreover, path models resemble network models with directed associations between variables (i.e. directed edges; Epskamp et al., 2017). Furthermore, Epskamp, Maris, Waldorp and Borsboom (2018) have shown that network models based on dichotomous data, estimated as Ising models, can reveal probability distributions that are statistically equivalent to the distribution of latent variable models (i.e. multidimensional Item Response Theory (MIRT) models).

Accordingly, in specific situations, network models can be equivalent to latent variable models and comparing methodologies of network models and (at the least) similar statistical models is thus not sufficient to justify our methodological choice. Yet, I believe that network models have several practical, as well as theoretical advantages for our research aim (‘to estimate and compare the interrelated system of RFs for groups of adolescents with and without a history of adversity’). First of all, network models (as opposed to other methods such as SEM) not only can estimate many variable interrelations (or in our case factor score interrelations) at the same time, but can also visualize those in form of a network graph. In my opinion, those graphs are highly insightful, as they enable the reader to process the interrelation strength and connectivity patterns of many RFs at the same time. Second, network

analysis also enables the calculation of coefficients that indicate which variables are most central (e.g. most strongly interrelated with other variables) in the model, which can be seen as straightforward summary metrics that supplement the network graphs. Notably, those interrelatedness (or 'centrality') coefficients are usually not established and/or facilitated with SEM or path models. Third, and potentially most importantly, I believe that the underlying theory that nurtures our models – namely that RFs, which are empirically found to help prevent psychopathological distress after adversity, may not necessarily function in isolation, but may function as a complex interrelated system – goes along well with the theory of network modelling. Generally, network modelling puts the focus on the studied variables (or nodes) themselves (Bringmann & Eronen, 2018), in our case the RFs, whereas for example latent variable models put the focus on an underlying latent concept that explains or is explained by the studied variables (Bringmann & Eronen, 2018), e.g. such as an overall score for resilient functioning. Therefore, I think that network modelling facilitates the analysis of our specific research aim ('to estimate the interrelated system of RFs for groups of adolescents with and without a history of adversity (i.e. '*exploratory*') and to compare the two group networks with each other (i.e. '*confirmatory*')), particularly well. Last, I would like to highlight that our research includes several methodological techniques in addition to network modelling, which are all chosen based on the specific analysis goal. I.e. our CA variable is derived from a latent class analysis, our RFs are estimated with confirmatory factor analyses, and our general distress variable is estimated with a bifactor model.

## APPENDIX B.2: RELIABILITY AND/OR VALIDITY INFORMATION FOR THE USED MEASURES

*Reliability and/or validity information for the used measures*

| Name Variable | (Sub-)Scale/ Interview Used | Validity/ Reliability |
|---|---|---|
| Childhood adversity | Cambridge Early Experiences Interview (CAMEEI; Dunn et al., 2011) | • CAMEEI (Dunn et al., 2011); inter-rater reliability kappa = 0.7 - 0.9 |
| General distress | Mood and Feeling Questionnaire (MFQ; 33 items; Messer, Angold, & Costello, 1995) + Revised Children's Manifest Anxiety Scale (RCMAS; 28 items; Reynolds & Richmond, 1978) | • MFQ (Wood, Kroll, Moore, & Harrington, 1995); internal consistency alpha = .94<br>• RCMAS (Reynolds & Richmond, 1978); $KR_{20}$ reliability = .85 |
| Friendship support | Cambridge Friendships Questionnaire (CFQ; 5 of 8 items used; Goodyer, Wright, & Altham, 1989) | • CFQ (van Harmelen et al., 2016); 2 weeks test retest reliability kappa = .80 |
| Family support | General Functioning subscale of the McMaster Family Assessment Device (GF-FAD; 5 of 12 items used; Epstein, Baldwin, & Bishop, 1983) | • GF-FAD (Epstein et al., 1983); Cronbach's alpha = 0.92 |
| Family cohesion | General Functioning subscale of the McMaster Family Assessment Device (GF-FAD; 7 of 12 items used; Epstein et al., 1983) | • GF-FAD (Epstein et al., 1983); Cronbach's alpha = 0.92 |
| Positive self-esteem | Rosenberg self-esteem scale (RSES; 5 of 10 items used; Rosenberg, 1965) | • RSES (Gray-Little, Williams, & Hancock, 1997); Cronbach's alpha = 0.88 |
| Negative self-esteem | Rosenberg self-esteem scale (RSES; 5 of 10 items used; Rosenberg, 1965) | • RSES (Gray-Little et al., 1997); Cronbach's alpha = 0.88 |
| Reflective rumination | Ruminative Response Scale (RRS; 5 of 22 items used; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) | • RRS (Treynor et al., 2003); Cronbach's alpha = .90;<br>• RRS 5 item subscale (Treynor et al., 2003); Cronbach's alpha = .72 |
| Ruminative brooding | Ruminative Response Scale (RSS; 5 of 22 items used; Treynor et al., 2003) + Short Leyton Obsessional Inventory (LOI; 2 of 11 items used; Bamber, Tamplin, Park, Kyte, & Goodyer, 2002) | • RRS (Treynor et al., 2003); Cronbach's alpha = .90;<br>• RRS 5 item subscale (Treynor et al., 2003); Cronbach's alpha = .77<br>• LOI (Bamber et al., 2002); internal reliability alpha = .94 |
| Distress tolerance | Emotionality subscale of the Emotionality Activity Sociability Temperament Survey (EAS; 5 items; Bould, Joinson, Sterne, & Araya, 2013) | • EAS emotionality subscale (Bould et al., 2013); Cronbach's alpha = .84 - 85 |
| Aggression | Behaviour Checklist (BC; 4 of 11 items; Goodyer et al., 2011) | • BC (Kiddle et al., 2017); Cronbach's alpha = .74 |
| Expressive suppression | Callous-unemotional subscale of the Antisocial Process Screening Device (CU-APSD; 1 of 6 items; Poythress et al., 2006) | • CU-APSD (Poythress et al., 2006); Cronbach's alpha = .22 - .60, with a median of .46 |

## APPENDIX B.3: VARIABLE PREPARATION

The results of the polychoric confirmatory factor analyses (CFAs) for the RFs can be found in Table B.1. We used the resulting latent factor scores of the RFs (i.e. standardized scores) as variables in the RF networks. We included recommendations from modification indices only if the suggestion could be theoretically underpinned, i.e. only if the suggested covariance was based on two similar worded items. Moreover, when items or item covariances led to negative (residual) variances, the respective item/covariance was removed from the CFA. This was done, as for models with negative (residual) variances factor scores cannot be established. For expressive suppression we used a scaled item score as variable (*n* = 1146), because expressive suppression was based on a single item.

The one-factor CFA for self-esteem (Rosenberg, 1965) revealed a poor fit, even after the addition of two item covariances (Robust CFI = 0.96, Robust TLI = 0.94, Robust SRMR = 0.07, Robust RMSEA = 0.15, RMSEA 90% CI = 0.14 – 0.15). Based on prior research we established a two factor CFA model, resulting in a positive and a negative self-esteem factor (Tomas & Oliver, 1999). Importantly, in a multiple-factor CFA we could not allow for covariances between factors. Allowing covariances between factors leads to inter-dependent factor scores. However, variables in networks cannot be based on inter-dependent scores, given that the aim of network analysis is to scrutinize the interrelation of variables and scrutinizing the interrelation of inter-dependent variables would be double dipping. Therefore, we established two one-factor models for positive and negative self-esteem, albeit being aware that the two models measure topologically similar concepts (Tomas & Oliver, 1999).

Based on Treynor and colleagues' (2003) findings, we excluded 12 of the 22 RRS (i.e. rumination) items that overlapped with validated depression items (i.e. items of the Beck Depression Inventory; Beck, Steer, & Garbin, 1988; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961) and utilized two separate rumination factors. Respectively, one rumination factor for brooding (Bamber et al., 2002; Treynor et al., 2003) and one for reflection (Treynor et al., 2003). For the same reason as for self-esteem, we established two one-factor CFAs for rumination.

Box-and-whisker plots with individual data points for the RFs (except expressive suppression) and the general distress variable can be found in Figure B.1. Location and dispersion values for the RFs and the general distress variable can be found in Table B.2. Due to the lack of variability we dichotomized aggression and expressive suppression RFs. Due to deviations from normality for some of the remaining eight RFs, we transformed these eight factor scores and the general distress variable using the nonparanormal transformation (Zhao, Liu, Roeder, Lafferty, & Wasserman, 2012).

Table B.1
*Polychoric confirmatory factor analyses conducted with the WLSMV estimator*

| Robust CFI | Robust TLI | Robust SRMR | Robust RMSEA | RMSEA 90% CI |
|---|---|---|---|---|
| **Friendship support (Goodyer et al., 1989), *1 factor, 5 items, 1 additional item covariance, n = 1138*** | | | | |
| **0.99** | 0.99 | 0.03 | 0.07 | 0.05 – 0.10 |
| **Family support (Epstein et al., 1983), *1 factor, 5 items, 1 additional item covariance, n = 1122*** | | | | |
| **1.00** | 0.99 | 0.02 | 0.08 | 0.05 – 0.10 |
| **Family cohesion (Epstein et al., 1983), *1 factor, 7 items, 1 additional item covariance, n = 1129*** | | | | |
| **0.98** | 0.97 | 0.04 | 0.08 | 0.07 – 0.10 |
| **Positive self-esteem (Rosenberg, 1965), *1 factor, 5 items, 1 additional item covariance, n = 1148*** | | | | |
| **1.00** | 0.99 | 0.01 | 0.08 | 0.06 – 0.11 |

| | | | | |
|---|---|---|---|---|
| **Negative self-esteem (Rosenberg, 1965), *1 factor, 5 items, 0 additional item covariances, n = 1151*** | | | | |
| **1.00** | 1.00 | 0.01 | 0.04 | 0.02 – 0.07 |
| **Rumination: Brooding (Bamber et al., 2002; Treynor et al., 2003), *1 factors, 7 items, 1 additional item covariance, n = 1139*** | | | | |
| **0.99** | 0.98 | 0.03 | 0.06 | 0.05 – 0.08 |
| **Rumination: Reflection (Treynor et al., 2003), *1 factor, 5 items, 1 additional item covariance, n = 1148*** | | | | |
| **1.00** | 1.00 | 0.01 | 0.02 | 0.00 – 0.06 |
| **Distress tolerance (Bould et al., 2013), *1 factor, 5 items, 1 additional item covariance, n = 1149*** | | | | |
| **0.98** | 0.96 | 0.04 | 0.14 | 0.12 – 0.17 |
| **Aggression (Goodyer et al., 2011), *1 factor, 4 items, 0 additional item covariances, n = 1156*** | | | | |
| **1.00** | 0.99 | 0.03 | 0.03 | 0.00 – 0.07 |

*Note.* WLSMV = weighted least squares estimator with mean- and variance corrected test statistics and robust standard errors. CFI = Comparative fit index, TLI = Tucker-Lewis index, SRMR = Standardized root mean square residual, RMSEA = Root mean square error of approximation, CI = Confidence interval.

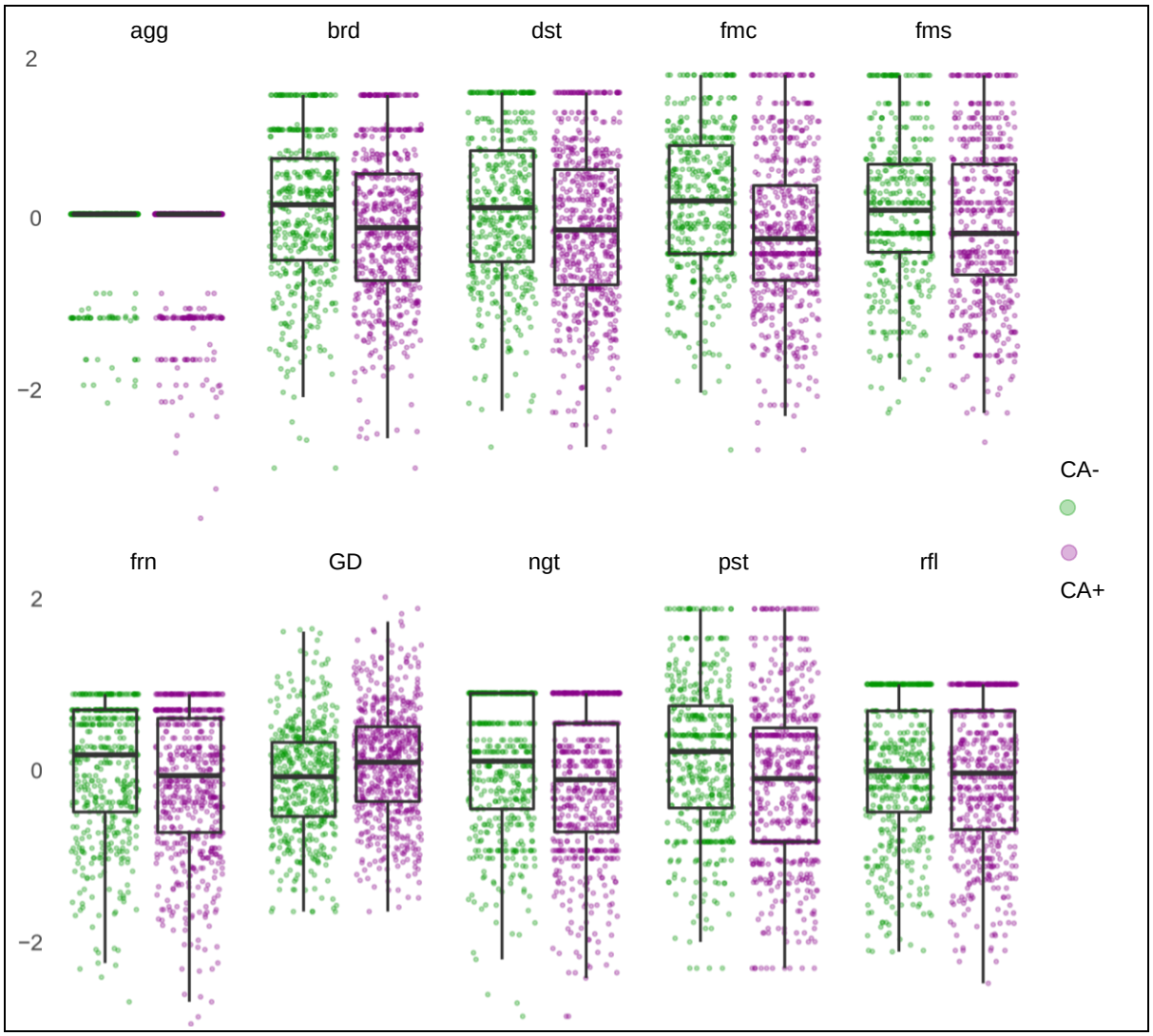



*Figure B.1.* Box-and-whisker plots with individual data points for the untransformed RFs (except expressive suppression) and the general distress variable, separately for CA+ (*n* = 638) and CA- (*n* = 501) groups. As expressive suppression contained three ordered categories (CA+: 1 = 26, 2 = 183, 3 = 408; CA-: 1 = 12, 2 = 117, 3 = 366) we considered box-and-whisker plots with individual data points as inappropriate. CA- group = green individual data points, CA+ group = magenta individual data points. Center line = median (50% quantile); lower box limit =25% quantile; upper box limit = 75% quantile; lower whisker = smallest observation greater than or equal to the lower box limit - 1.5 x Inter Quartile Range (IQR); upper whisker = largest observation less than or equal to upper box limit + 1.5 x IQR; outliers = data points beyond the end of the whiskers. **Legend:** Agg = aggression, brd = brooding, dst = distress tolerance, fmc = family cohesion, fms = family support, frn = friend support, ngt = negative self-esteem, GD = general distress, pst = positive self-esteem, rfl = reflective rumination.

Table B.2
*Means and standard deviations or frequencies for untransformed RF and the general distress variables of CA+ (n = 638) and CA- (n = 501) groups*

| | ***CA+*** | | ***CA-*** | |
|---|---|---|---|---|
| **Variable[*1/*2/*3]** | ***N*** | **Mean(SD) / Median(IQR)** | ***N*** | **Mean(SD) / Median(IQR)** |
| Friendship support (high) | 606 | -0.13 (.82) | 480 | -0.01 (.76) |
| Family support (high) | 585 | -0.07 (.91) | 481 | 0.08 (.85) |
| Family cohesion (high) | 585 | -0.16 (.90) | 488 | 0.18 (.81) |
| Negative self-esteem (low) | 610 | -0.14 (.84) | 488 | 0.05 (.77) |
| Positive self-esteem (high) | 611 | -0.12 (.95) | 486 | 0.16 (.88) |
| Brooding (low) | 604 | -0.09 (.89) | 486 | 0.07 (.87) |
| Reflective rumination (low) | 608 | -0.07 (.84) | 487 | -0.01 (.82) |
| Distress tolerance (high) | 618 | -0.12 (.91) | 494 | 0.12 (.87) |
| Aggression (low) | 613 | -0.24 (.61) | 491 | -0.11 (.44) |
| Expressive suppression (low) | 617 | 0.63 (1.86) | 495 | 0.63 (1.86) |
| General distress | 616 | 0.08 (.65) | 490 | -0.10 (.65) |

*Note.* CA = childhood adversity, SD = standard deviation, IQR = inter quartile range. [*1]All RFs are scored in such a way that high values are protective (e.g. high levels of high friendship support or high levels of low negative self-esteem) and low values are harmful (e.g. low levels of high friendship support or low levels of low negative self-esteem). [*2]The continuous general distress variable is scored in such a way that the higher the value the higher the level of general distress. [*3]As expressive suppression contained three ordered categories we calculated the median and the inter quartile range, for all other variables the mean and the standard deviation were calculated.

## APPENDIX B.4: ASSOCIATION NETWORK FOR CA+ AND CA- GROUPS

For the CA- group, the association network (i.e. zero-order correlations; see Figure B.2) showed that all RFs are positively correlated, except for the two relationships between expressive suppression and distress tolerance as well as expressive suppression and positive self-esteem. Interestingly, in the association network of the CA+ group (see Figure B.2), expressive suppression was negatively associated with distress tolerance, reflective rumination, friendship support, and brooding.

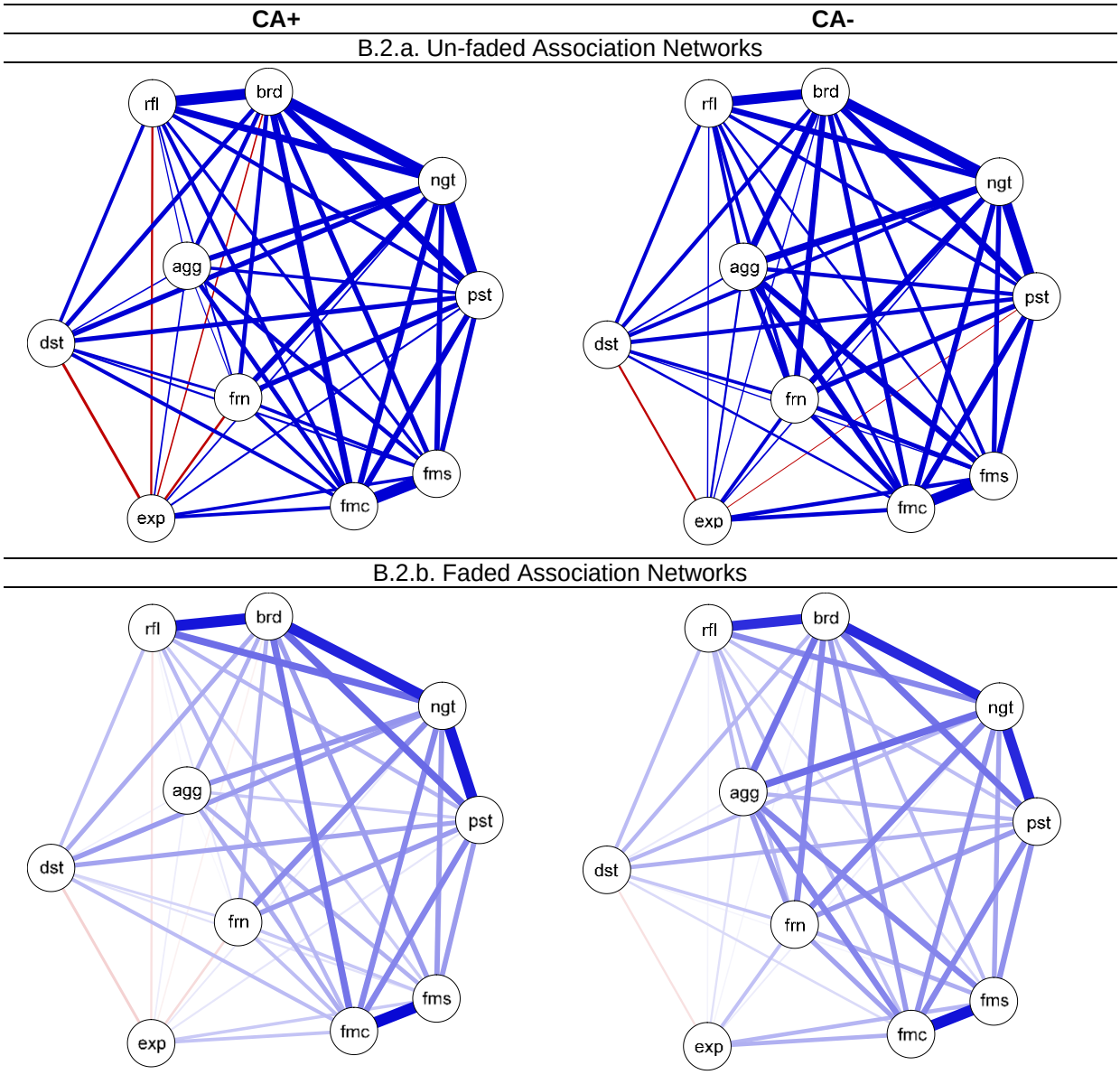



*Figure B.2.* Association network for the CA+ (*n* = 638) and the CA- (*n* = 501) group. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflective rumination, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression.

## APPENDIX B.5: INTERCONNECTEDNESS OF RESILIENCE FACTORS

In both the CA+ and the CA- regularized partial correlation (reg-pcor) networks there were particularly strong positive relationships between high family cohesion and high family support (reg-pcor CA+ = .59, CA- = .55), low brooding and low reflective rumination (reg-pcor CA+ = .51, CA- = .45), low negative and high positive self-esteem (reg-pcor CA+ = .46, CA- = .42), and between low brooding and low negative self-esteem (reg-pcor CA+ = .36, CA- = .36). Interestingly, low expressive suppression was associated with *high* positive self-esteem and *low* friendship support in the CA+ network, which was reversed in the CA- network (i.e. low expressive suppression with *low* positive self-esteem and *high* friendship support). Furthermore, in the CA+ network low aggression was associated with *low* friendship support, whereas the opposite pattern was revealed in the CA- network (i.e. low aggression with *high* friendship support). To examine the interrelatedness of the RFs, we calculated three coefficients. *Node strength* is the sum of the interrelation values (e.g. regularized partial correlations) of a given RF with all directly related RFs (i.e. the sum of the absolute values of the RF interrelations; Costantini et al., 2015; McNally, 2016). *Expected influence* is based on the formula of node strength, but takes negative relationships between RFs into account (i.e. the sum of the relative values of the RF interrelations; McNally, 2016). *Node predictability* is defined as the amount of variance of each RF that is explained by the directly related RFs (i.e. absolute metric ranging from zero to 100 percent explained variance; Haslbeck & Waldorp, 2018). Node strength, expected influence and predictability had very similar RF importance rankings (Table B.3). In sum, the self-esteem, brooding, and family RFs had the highest strength, expected influence and predictability values. Interestingly, low expressive suppression had a negative expected influence coefficient for the CA+ group (-0.06), but a positive coefficient for the CA- group (0.17).

Table B.3

*Node strength (S), expected influence (EI), node predictability (P), and the belonging coefficient rank in parenthesis, for networks without the general distress variable*

| CA | negative SE | brooding | family cohesion | positive SE | family support | reflection | aggression | friendship support | expressive suppression | distress tolerance |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Strength | | | | | |
| | *1.* | *2.* | *3.* | *4.* | *5.* | *6.* | *7.* | *8.* | *9.* | *10.* |
| yes | 1.25 | 1.10 | 1.04 | 0.76 | 0.76 | 0.66 | 0.38 | 0.37 | 0.37 | 0.37 |
| no | 1.06 | 1.14 | 0.94 | 0.78 | 0.83 | 0.53 | 0.59 | 0.58 | 0.34 | 0.34 |
| | *2.* | *1.* | *3.* | *5.* | *4.* | *8.* | *6.* | *7.* | *9.* | *10.* |
| | | | | | Expected Influence | | | | | |
| | *1.* | *2.* | *3.* | *4.* | *5.* | *6.* | *7.* | *8.* | *10.* | *9.* |
| yes | 1.25 | 1.10 | 1.04 | 0.76 | 0.76 | 0.45 | 0.23 | 0.19 | -0.06 | 0.17 |
| no | 1.06 | 1.14 | 0.94 | 0.73 | 0.83 | 0.53 | 0.59 | 0.58 | 0.17 | 0.22 |
| | *2.* | *1.* | *3.* | *5.* | *4.* | *8.* | *6.* | *7.* | *10.* | *9.* |
| | | | | | Predictability | | | | | |
| | *2.* | *1.* | *3.* | *6.* | *4.* | *5.* | *10.* | *7.* | *9.* | *8.* |
| yes | 0.57 | 0.61 | 0.54 | 0.42 | 0.48 | 0.44 | 0.00 | 0.14 | 0.07 | 0.11 |
| no | 0.52 | 0.53 | 0.48 | 0.42 | 0.47 | 0.34 | 0.02 | 0.18 | 0.02 | 0.08 |
| | *2.* | *1.* | *3.* | *5.* | *4.* | *6.* | *9.* | *7.* | *10.* | *8.* |

*Note.* CA = Childhood adversity (yes: *n* = 638, no: *n* = 501). SE = Self-esteem.

## APPENDIX B.6: RESILIENCE FACTOR INTERRELATEDNESS COEFFICIENTS BASED ON NETWORKS CORRECTED FOR THE GENERAL DISTRESS VARIABLE

Node strength and expected influence coefficients changed slightly in both groups, when taking general distress levels into account (correlation between the 10 RF coefficients of the networks without the distress variable and the networks corrected for the variance of the distress variable; *node strength*: CA+ r = .75, CA- r = .79; *expected influence*: CA+ r = .93, CA- r = .84). Importantly, the coefficient ranks changed notably after correcting for the general distress variable (see change in the coefficient rank order from Table B.3 to Table B.4).

Table B.4

*Node strength (S), expected influence (EI), and the belonging coefficient rank in parenthesis, for networks corrected for the general distress variable*

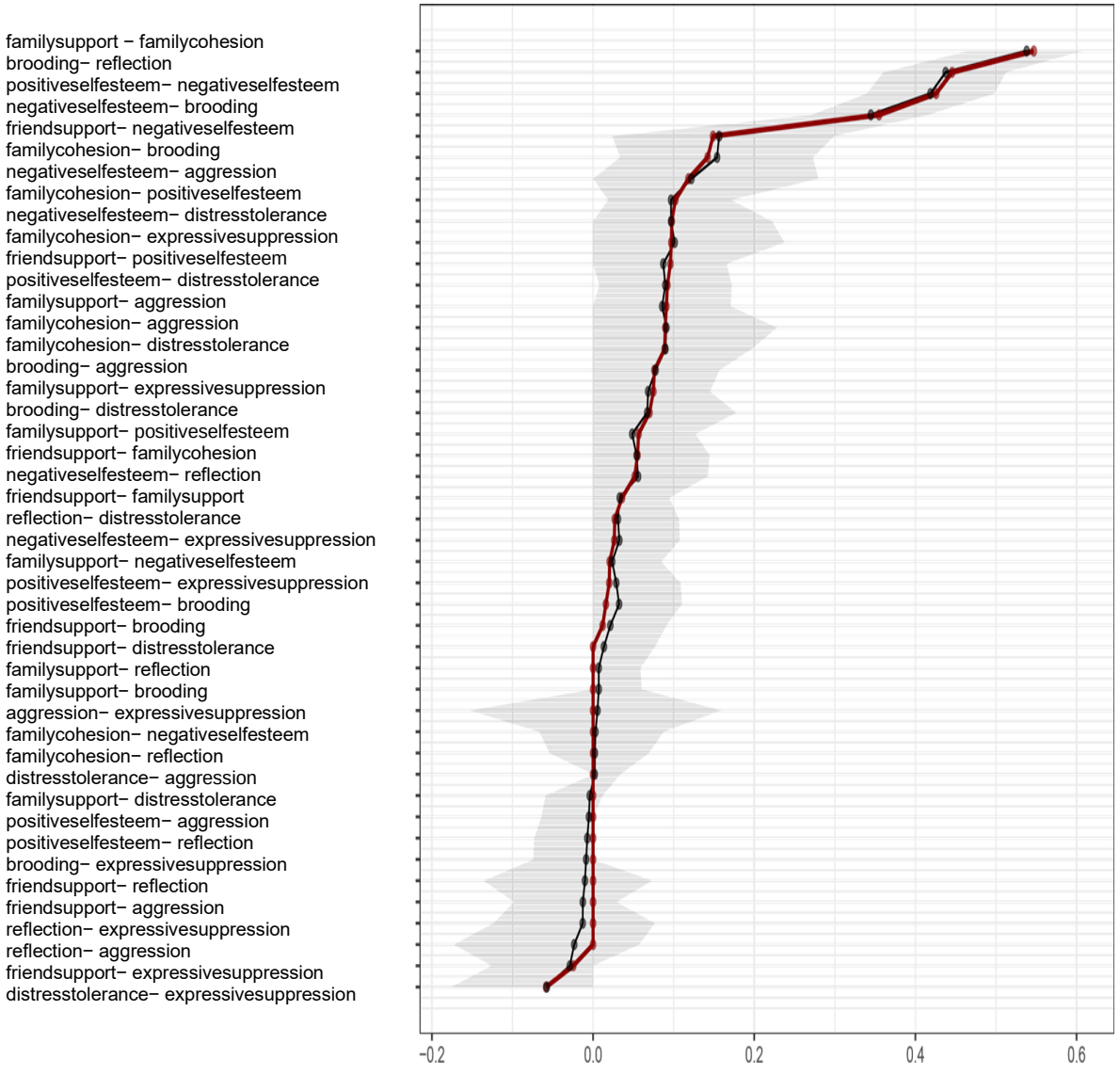
| **CA** | **negative SE** | **brooding** | **family cohesion** | **positive SE** | **family support** | **reflection** | **aggression** | **friendship support** | **expressive suppression** | **distress tolerance** |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Strength | | | | | |
| | 3. | 6. | 1. | 5. | 4. | 2. | 7. | 9. | 8. | 10. |
| yes | 0.91 | 0.75 | 1.04 | 0.80 | 0.84 | 0.99 | 0.61 | 0.51 | 0.61 | 0.44 |
| no | 0.57 | 0.65 | 0.83 | 0.62 | 0.81 | 0.43 | 0.31 | 0.47 | 0.30 | 0.28 |
| | *5.* | *3.* | *1.* | *4.* | *2.* | *7.* | *8.* | *6.* | *9.* | *10.* |
| | | | | | Expected Influence | | | | | |
| | *2.* | *4.* | *1.* | *5.* | *3.* | *6.* | *10.* | *9.* | *8.* | *7.* |
| yes | 0.86 | 0.72 | 0.99 | 0.46 | 0.76 | 0.18 | -0.19 | -0.17 | -0.03 | 0.01 |
| no | 0.57 | 0.65 | 0.83 | 0.60 | 0.81 | 0.43 | 0.31 | 0.47 | 0.17 | 0.18 |
| | *5.* | *3.* | *1.* | *4.* | *2.* | *7.* | *8.* | *6.* | *10.* | *9.* |

*Note.* CA = Childhood adversity (yes: *n* = 638, no: *n* = 501). SE = Self-esteem.

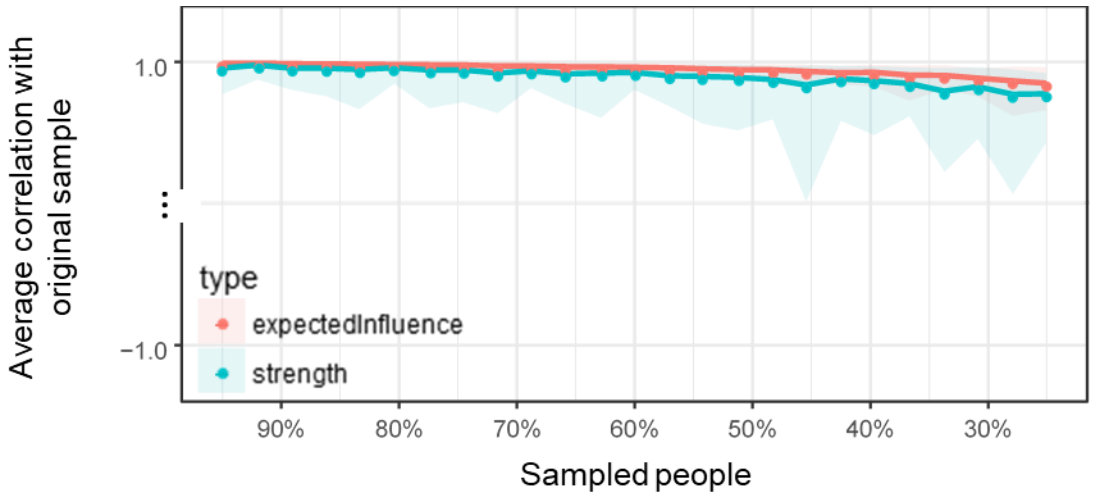
## APPENDIX B.7: ROBUSTNESS ANALYSES: ACCURACY AND STABILITY OF THE RESILIENCE FACTOR NETWORK MODELS

To test the accuracy of the regularized partial correlation RF models we bootstrapped the RF interrelations (*N* boot = 2000) and to test the stability of the *node strength* and *expected influence* coefficients we applied a subset bootstrap (*N* boot = 2000). For CA+ and CA- groups, family support and family cohesion had the highest interrelation, followed by reflective rumination and brooding, negative and positive self-esteem, as well as by negative self-esteem and brooding (Figure B.3). Additional analyses showed that these four RF interrelations differed significantly from all other RF interrelations. The bootstrapped interrelation CIs had an acceptable width and we concluded that our models had a sufficient RF interrelation accuracy. With regard to the *node strength* and *expected influence* stability, we found for the CA+ network that up to 74.9 percent of the sample could be dropped

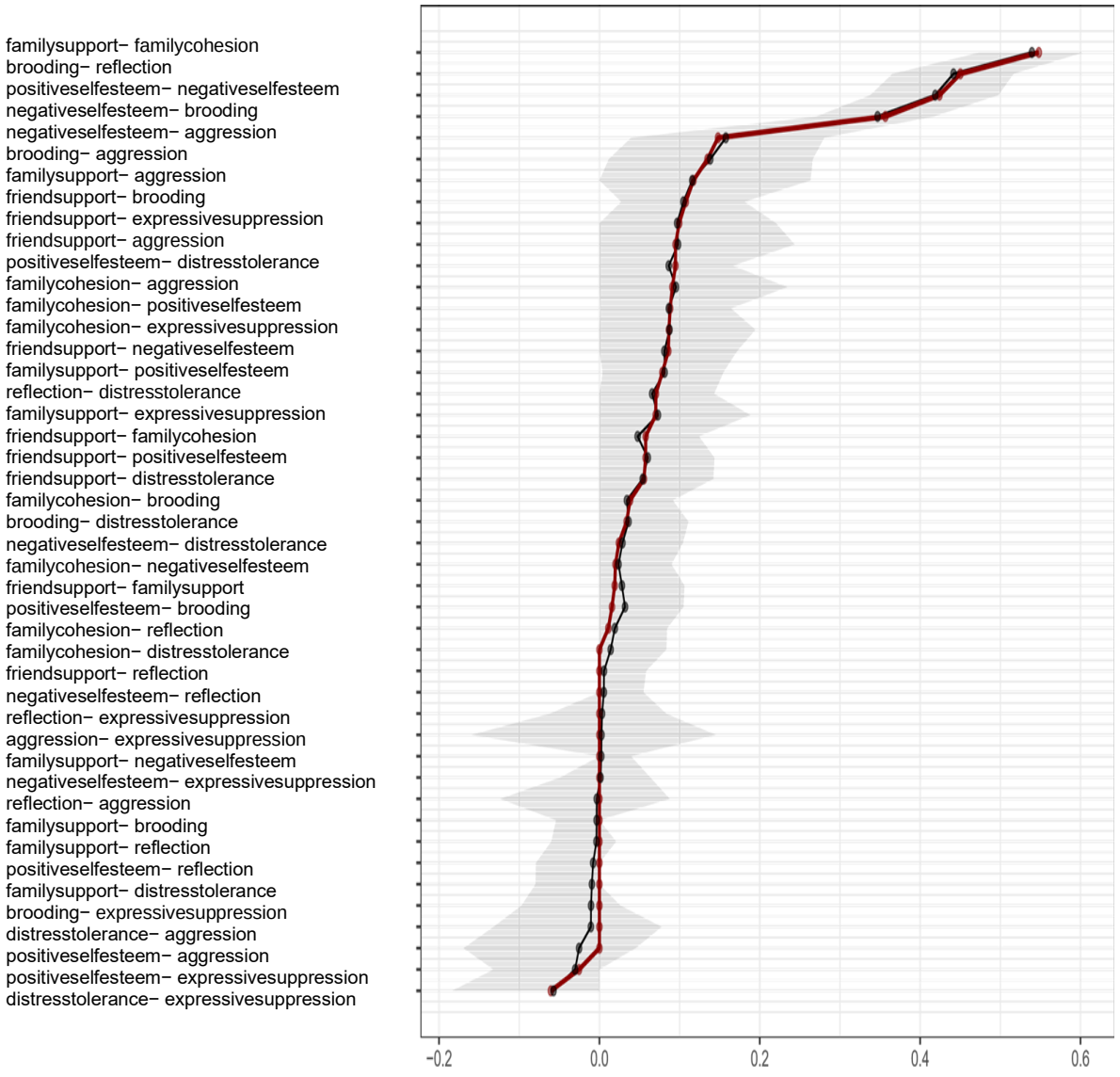
B.3.a. RF Interrelation Accuracy for the CA+ Group



B.3.b. Node Strength and Expected Influence Stability for the CA+ Group




B.3.c. RF Interrelation Accuracy for the CA- Group



B.3.d. Node Strength and Expected Influence Stability for the CA- Group

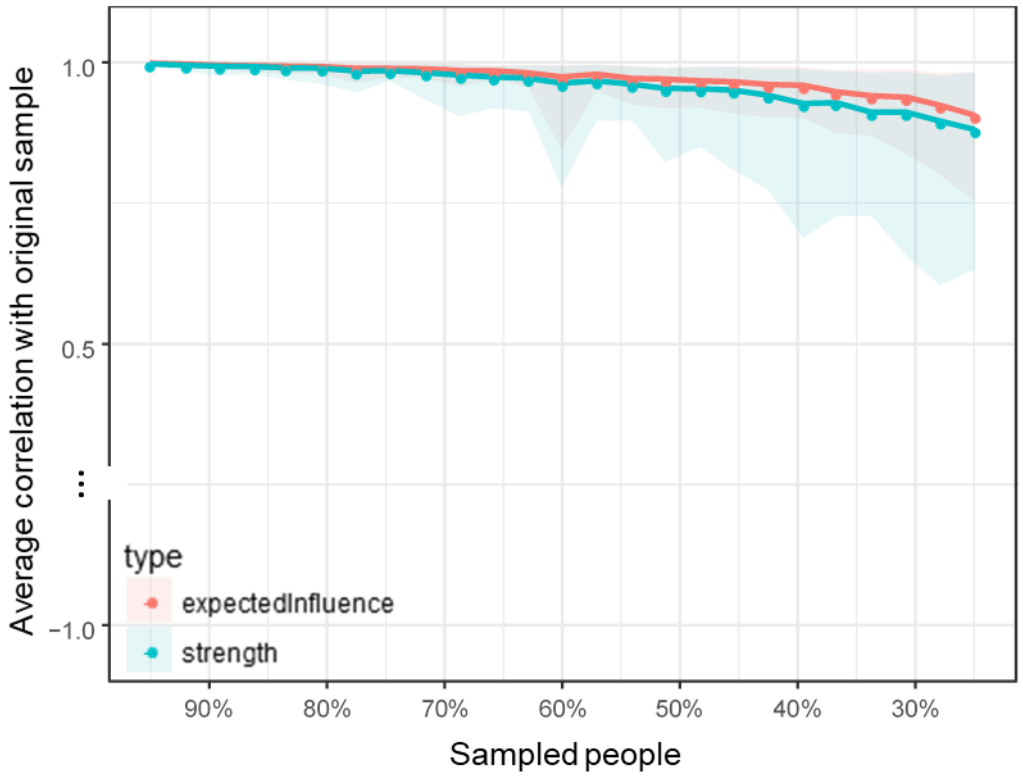



*Figure B.3.* RF interrelation accuracy (3.a. and 3.c.) and node strength and expected influence stability (3.b. and 3.d.) plots for CA+ and CA- groups. Panel '3.a.' and '3.b.' depict the CA+ (*n* = 638) and panel '3.c.' and '3.d.' the CA- plots (*n* = 501). Panel '3.a.' and '3.c.' depict the sample RF interrelations (i.e. edge weights) which are represented by the red dots, the means of the bootstrapped RF interrelations (i.e. edge weights) which are represented by the black dots, and the belonging bootstrap confidence intervals (CIs) which indicate the RF interrelation accuracy. Panel '3.b.' and '3.d.' depict the average correlation of the node strength (and expected influence) coefficients between the original sample and the sample subsets.

to reveal (with a 95 percent likelihood) an association of minimal 0.7 between the subset and the original *node strength* (or *expected influence*) coefficients. This subset dropping percentage, of both *node strength* and *expected influence*, was 75 for the CA- network. Therefore, we concluded that our models had a sufficient stability of the *node strength* and *expected influence* coefficients.

## APPENDIX B.8: SENSITIVITY ANALYSES: STATISTICAL SOUNDNESS OF THE RESILIENCE FACTOR NETWORK MODLES

To allow for the largest possible sample size we based the network models on the full-information sample, using all possible pairwise correlations. This led to the result that different RF interrelation coefficients are based on different sample sizes. To substantiate the feasibility of this approach, we tested the extent to which the RF interrelations of the full-information (*N* CA+ = 638; *N* CA- = 501) and the complete-information (*N* CA+ = 508; *N* CA- = 443) samples are associated with each other. For both the CA+ and the CA- group, the two regularized partial correlation networks were highly correlated (adjacency matrix correlation for CA+: r = 0.99; for CA-: r = 0.997). Similarly, the RF predictability networks (i.e. those models are not discussed in the text, but were established for the calculation of the predictability coefficients) had to be based on the complete-information subsets of the two samples (CA+ and CA-). Therefore, we also scrutinized the relationship between the RF interrelations of the full-information regularized partial correlation networks and the predictability networks. Those RF interrelations were also highly correlated (adjacency matrix correlation for CA+: r = 0.94; for CA-: r = 0.97), indicating similarity between the results of the two methods.

Given that we pre-processed the RF variables through establishing factor scores and through applying transformations (i.e. nonparanormal method), we additionally performed sensitivity analyses to test the similarity of the reported regularized partial correlation networks with networks using (1) factor scores without transformation, (2) mean scores with transformation, and (3) mean scores without transformation. As all three additional models correlated highly with our reported models for the CA+ and the CA- groups (which were based on factor scores with transformation), we concluded that our results are robust for the scrutinized sample (Table B.5).

Table B.5
*Sensitivity analysis for the regularized partial correlation network models being based on factor scores and making use of the nonparanormal transformation*

| | | **Reference model: factor scores, with transformation; comparison model:** | | |
|---|---|---|---|---|
| **CA** | **correlation type** | **factor scores, no transformation** | **mean scores, with transformation** | **mean scores, no transformation** |
| yes | Pearson | .999 | .99 | .99 |
| | Spearman | .99 | .97 | .97 |
| no | Pearson | .999 | .99 | .99 |
| | Spearman | .995 | .95 | .92 |

*Note.* CA = Childhood adversity (yes: *n* = 638, no: *n* = 501).

## APPENDIX B.9: INDIVIDUAL RESILIENCE FACTOR INTERRELATION DIFFERENCES BETWEEN THE CA+ AND THE CA- NETWORKS

Significant (and marginally significant) differences between individual RF interrelations of the CA+ and the CA- networks (i.e. compared to the same individual RF interrelation differences between permuted network model pairs), before and after Holm-Bonferroni correction (see Table B.6).

Table B.6
*Individual RF interrelation differences between the CA+ and the CA- networks*

| Name RF1 | Name RF2 | Difference estimate | p-value |
|---|---|---|---|
| **RF Networks** | | | |
| *After correction* | | | |
| Friendship support | Expressive suppression | 0.17 | <.01 |
| *Before correction* | | | |
| Friendship support | Brooding | 0.11 | .03 |
| Friendship support | Aggression | 0.11 | .04 |
| Friendship support | Expressive suppression | 0.17 | <.001 |
| Family cohesion | Brooding | 0.11 | <.01 |
| Positive self-esteem | Expressive suppression | 0.04 | .07 |
| **RF Networks with the General Distress Variable** | | | |
| *After correction* | | | |
| Friendship support | Reflection | 0.11 | .09 |
| Friendship support | Expressive suppression | 0.20 | <.001 |
| *Before correction* | | | |
| Friendship support | Brooding | 0.08 | <.01 |
| Friendship support | Reflection | 0.11 | <.01 |
| Friendship support | Aggression | 0.20 | <.01 |
| Friendship support | Expressive suppression | 0.20 | <.001 |
| Friendship support | General distress | 0.14 | <.01 |
| Family cohesion | Negative self-esteem | 0.03 | .04 |
| Family cohesion | Brooding | 0.09 | .05 |
| Family cohesion | Distress tolerance | 0.08 | .07 |
| Positive self-esteem | Reflection | 0.09 | .05 |
| Positive self-esteem | Expressive suppression | 0.05 | .05 |
| Negative self-esteem | Reflection | 0.07 | <.01 |
| Negative self-esteem | Expressive suppression | 0.09 | .02 |
| Brooding | Reflection | 0.12 | .02 |
| Reflection | Aggression | 0.15 | .04 |
| Reflection | Expressive suppression | 0.07 | .08 |
| Expressive suppression | General distress | 0.06 | .09 |
| **RF Networks Corrected for the General Distress Variable** | | | |
| *After correction* | | | |
| Friendship support | Reflection | 0.11 | .07 |
| Friendship support | Expressive suppression | 0.20 | <.01 |
| *Before correction* | | | |
| Friendship support | Brooding | 0.08 | <.01 |
| Friendship support | Reflection | 0.11 | <.01 |
| Friendship support | Aggression | 0.20 | <.01 |
| Friendship support | Expressive suppression | 0.20 | <.001 |
| Family cohesion | Negative self-esteem | 0.03 | .04 |
| Family cohesion | Brooding | 0.09 | .05 |
| Family cohesion | Distress tolerance | 0.08 | .07 |
| Positive self-esteem | Reflection | 0.09 | .04 |
| Positive self-esteem | Expressive suppression | 0.05 | .06 |
| Negative self-esteem | Reflection | 0.07 | <.01 |
| Negative self-esteem | Expressive suppression | 0.09 | .02 |
| Brooding | Reflection | 0.12 | .01 |
| Reflection | Aggression | 0.15 | .04 |
| Reflection | Expressive suppression | 0.07 | .08 |

## APPENDIX B.10: ADJACENCY MATRICES OF THE MAIN MODELS

| | friendship support | family support | family cohesion | positive SE | negative SE | brooding | reflection | distress tolerance | aggres-sion | exp. sup-press. |
|---|---|---|---|---|---|---|---|---|---|---|
| **1: Weights matrix for the lasso regularized RF network model of the CA+ group** | | | | | | | | | | |
| friendship support | - | .023909 | .028839 | .080385 | .148546 | .000000 | .000000 | .000000 | -.017070 | -.071317 |
| family support | | - | .587941 | .031853 | .017381 | .000000 | .000000 | .000000 | .058560 | .036042 |
| family cohesion | | | - | .088599 | .000000 | .144786 | .000000 | .050993 | .056685 | .085883 |
| positive SE | | | | - | .455981 | .009506 | .000000 | .078308 | .000000 | .013499 |
| negative SE | | | | | - | .355060 | .025007 | .088398 | .144010 | .017677 |
| brooding | | | | | | - | .512462 | .034055 | .046442 | .000000 |
| reflection | | | | | | | - | .018345 | -.058021 | -.046307 |
| distress tolerance | | | | | | | | - | .000000 | -.096202 |
| aggres-sion | | | | | | | | | - | .000000 |
| exp. sup-press. | | | | | | | | | | - |
| **2: Weights matrix for the lasso regularized RF network model of the CA- group** | | | | | | | | | | |
| friendship support | - | .019055 | .057729 | .057601 | .085972 | .107758 | .000000 | .055556 | .094934 | .099539 |
| family support | | - | .548010 | .078507 | .000000 | .000000 | .000000 | .000000 | .117245 | .070202 |
| family cohesion | | | - | .088205 | 0.020039 | .038283 | .011123 | .000000 | .091018 | .086695 |
| positive SE | | | | - | .424114 | .015679 | .000000 | .094866 | .000000 | -.025461 |
| negative SE | | | | | - | .356621 | .000000 | .024278 | .147978 | .000000 |
| brooding | | | | | | - | .450166 | .033913 | .134804 | .000000 |
| reflection | | | | | | | - | .070500 | .000000 | .000000 |
| distress tolerance | | | | | | | | - | .000000 | -.060563 |
| aggres-sion | | | | | | | | | - | .000000 |
| exp. sup-press. | | | | | | | | | | - |
| **3: Weights matrix for the lasso regularized RF network model of the CA+ group, including general distress** | | | | | | | | | | |
| GD | -.195287 | -.008303 | -.089868 | -.156796 | -.374769 | -.351656 | -.046490 | -.093833 | -.228264 | .062298 |
| friendship support | - | .038692 | .013288 | .041158 | .073642 | .000000 | -.106482 | .000000 | -.125764 | -.106961 |
| family support | | - | .606414 | .042512 | .000000 | .000000 | .000000 | -.041521 | .066804 | .041998 |
| family cohesion | | | - | .058362 | -.025743 | .097499 | .000000 | .082392 | .041399 | .118914 |
| positive SE | | | | - | .375004 | .000000 | -.084934 | .071999 | -.082701 | .039164 |
| negative SE | | | | | - | .134735 | .069420 | .050035 | .088657 | .089301 |
| brooding | | | | | | - | .493546 | .000000 | .012984 | -.014994 |
| reflection | | | | | | | - | .018847 | -.150017 | -.065218 |
| distress tolerance | | | | | | | | - | -.044511 | -.128313 |
| aggres-sion | | | | | | | | | - | .000000 |
| exp. sup-press. | | | | | | | | | | - |

**4: Weights matrix for the lasso regularized RF network model of the CA- group, including general distress**

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| GD | -.055698 | .000000 | -.065887 | -.096395 | -.402710 | -.366777 | -.070095 | -.020364 | -.235897 | .000000 |
| friendship support | - | .019879 | .054140 | .044121 | .059302 | .077595 | .000000 | .048046 | .075566 | .090010 |
| family support | | - | .542330 | .074108 | .000000 | .000000 | .000000 | .000000 | .111047 | .065862 |
| family cohesion | | | - | .073279 | .000000 | .010217 | .000000 | .000000 | .069631 | .081735 |
| positive SE | | | | - | .331164 | .000000 | .000000 | .086838 | .000000 | -.012585 |
| negative SE | | | | | - | .139278 | .000000 | .012990 | .026746 | .000000 |
| brooding | | | | | | - | .370515 | .024028 | .027801 | .000000 |
| reflection | | | | | | | - | .060800 | .000000 | .000000 |
| distress tolerance | | | | | | | | - | .000000 | -.051089 |
| aggres-sion | | | | | | | | | - | .000000 |
| exp. sup-press. | | | | | | | | | | - |

*Note.* CA = childhood adversity (yes: n = 638, no: n = 501); SE = self-esteem; Exp. suppress. = expressive suppression; GD = general distress.

## APPENDIX B.11: MAIN MODELS DEPICTED WITH FADED INTERRELATIONS

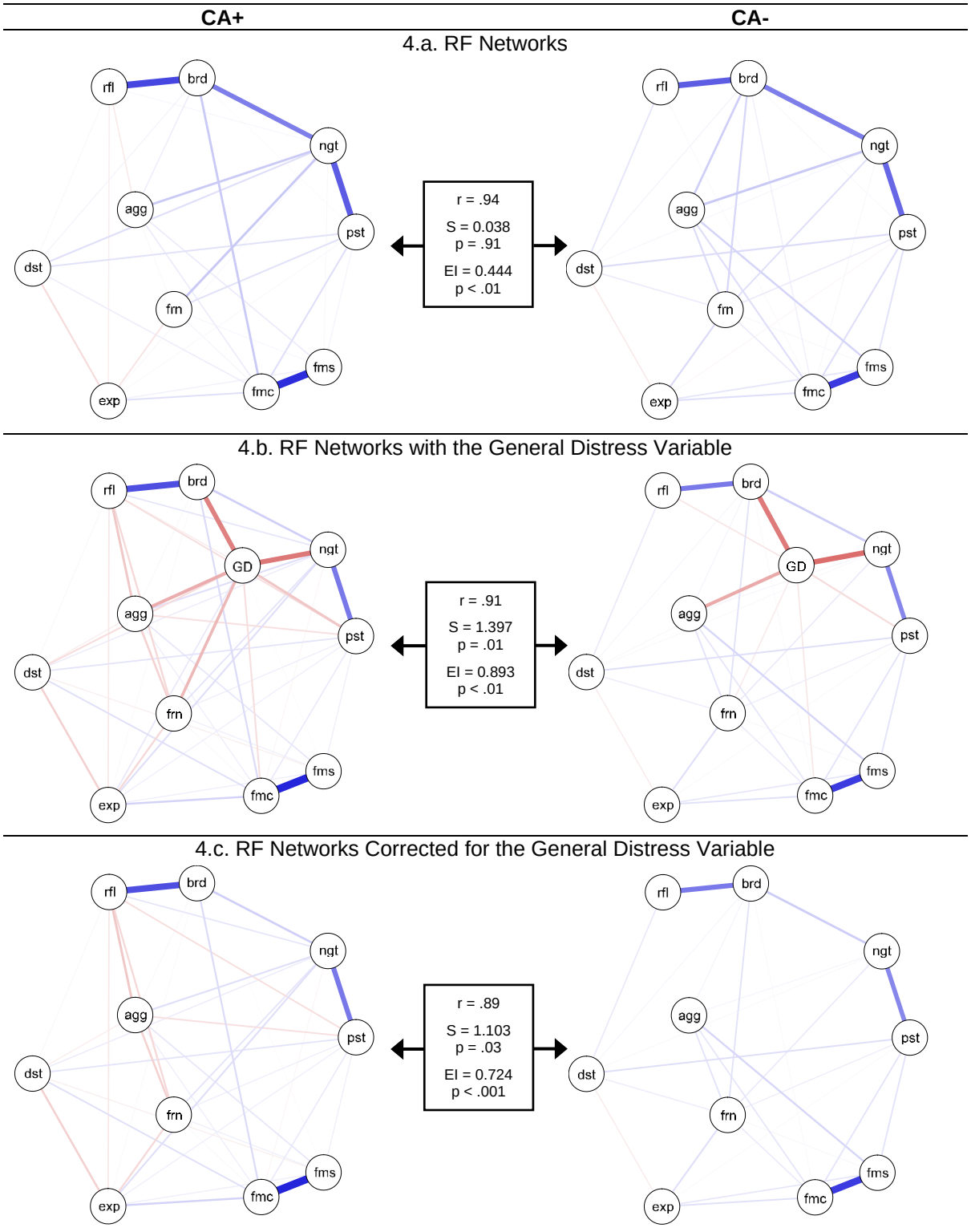



*Figure B.4.* CA+ (*n* = 638) and CA- (*n* = 501) resilience factor networks without (1.a.), with (1.b.), and corrected for (1.c.) the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the adjacency matrix correlation between the respective two networks (r), the difference in global network strength between the respective two networks (S), the difference in global network expected influence (EI) between the respective two networks (EI), and the p-value corresponding to the global network strength and global network EI comparisons (5000 comparison samples).

## APPENDIX B.12: EXPLORING THE INFLUENCE OF EXPRESSIVE SUPPRESSION ON THE RESILIENCE FACTOR NETWORKS OF EACH GROUP

For the CA- group the expressive suppression RF was only in the association (i.e. zero-order correlations), but not in the regularized partial correlation network negatively associated with the general distress variable. For the CA+ group the expressive suppression RF was positively related with the general distress variable in both the association (i.e. zero-order correlation) and the regularized partial correlation network (see main text Table 3). As, in the CA+ group, expressive suppression had a positive zero-order correlation (i.e. relationship which is not corrected for the impact of the other RFs) with general distress (shown in bold in main text Table 3.b.), the unexpected relationship sign is not the result of correcting for the other RFs.

Further exploratory analyses revealed that expressive suppression (in isolation) neither moderated (interaction effect: b = 0.10, SE = 0.13, t = 0.75, p = 0.45), nor mediated (indirect effect = -0.001, SE = 0.005, z = -0.22, p = .82, CI[-.012, .009]) the relationship between CA and the general distress variable. Hence, in our sample, expressive suppression did (cross-sectionally) not seem to function as RF.

Based on this finding and on the fact that expressive suppression was (in contrast to the other RFs) assessed with a single item, we re-estimated the regularized partial correlation RF networks for CA+ and CA- groups this time without the expressive suppression variable (Figure B.5). As in the models including expressive suppression, the relationship between aggression and friendship support was negative in the CA+ network, but positive in the CA- network. Moreover, both the CA+ and the CA- network revealed strong positive relationships between high family cohesion and high family support, low brooding and low reflective rumination, low negative and high positive self-esteem, as well as between low brooding and low negative self-esteem. Along those lines, the self-esteem, brooding, and family RFs had the highest strength and expected influence values. In sum, the RF networks without the expressive suppression variable resembled the corresponding networks including the variable.

The new regularized partial correlation networks of the CA+ and the CA- group were highly correlated (correlation between the 36 regularized RF interrelations of each group: r = 0.95). Moreover, the network structure invariance test was not significant ($M$ = .12, $N$ permutations = 5000, $p$ = 0.74), and the new CA+ and CA- networks did neither differ with regard to the global network strength ($S$ = 0.059, $S_{CA+}$ = 3.178, $S_{CA-}$ = 3.237, $N$ permutations = 5000, $p$ = 0.86), the global network expected influence (EI; $EI$ = 0.228, $EI_{CA+}$ = 3.009, $EI_{CA-}$ = 3.237, $N$ permutations = 5000, $p$ = 0.08), nor with regard to single interrelation differences (36 tests, Holm-Bonferroni corrected: $N$ permutations = 5000, corrected $p > 0.05$). As in the networks including expressive suppression, the degree of RF enhancement (i.e. 'global network EI') was higher in the CA- than in the CA+ network, yet, in the new networks this difference did not reach significance.

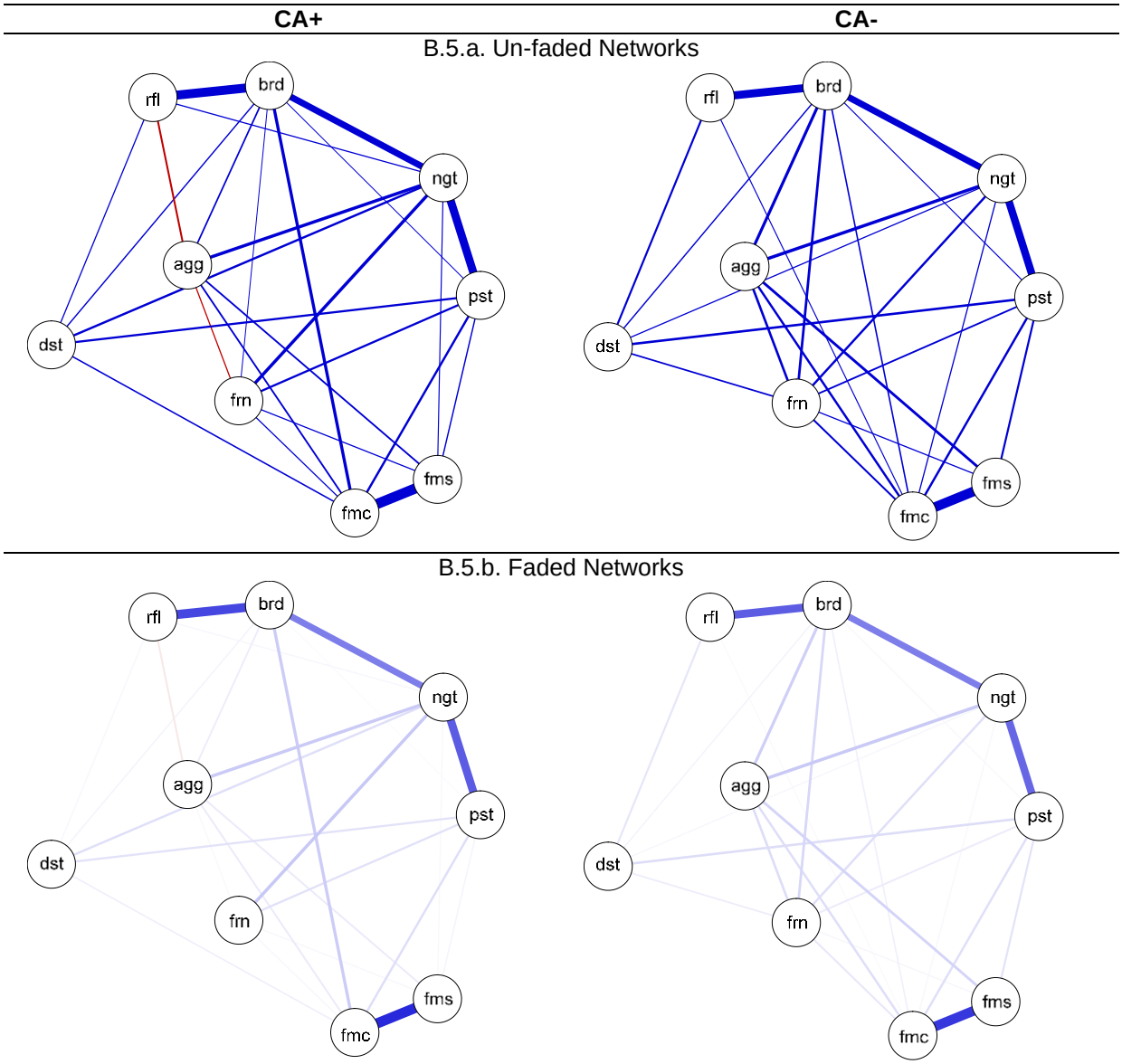



*Figure B.5.* Regularized partial correlation network without the expressive suppression variable for the CA+ (*n* = 638) and the CA- (*n* = 501) group. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflective rumination, brd = brooding, dst = distress tolerance, agg = aggression.

## APPENDIX B.13: NETWORK PATHWAYS BEWTEEN THE RESILIENCE FACTORS AND GENERAL DISTRESS

We investigated the Shortest Paths Lengths ('shortest pathways') between the RFs and the general distress variable, for both the CA+ and the CA- networks. The shortest pathway between two variables indicates the direct or indirect connection between those two variables along the strongest connection(s), or in other words the 'quickest' way between the two variables. Hence, shortest pathways designate whether the RFs have a direct connection with the general distress variable, or an indirect connection via other RFs. We found that the pathways between the RFs and the general distress variable differed for as many as 50 percent of the RFs. The five shortest pathways that differed between the CA+ and the CA- group can be seen in the main text Figure 2 and the five shortest pathways that were equivalent can be found in Figure B.6. In the CA+ group friendship support, family cohesion, and distress tolerance had direct shortest pathways with the general distress variable, whereas in the CA- group these shortest pathways went via intermediate RFs (see Figure 2 in main text). Moreover, the shortest pathway for family support and the general distress variable went in the CA+ group via family cohesion and in the CA- group via aggression. Similarly, the shortest pathway for expressive suppression and the general distress variable went in the CA+ group via negative self-esteem, and in the CA- group via friendship support and brooding. In both CA+ and CA- networks negative self-esteem, brooding and aggression had a direct shortest pathway with the general distress variable (see Figure B.6). Moreover, in both CA+ and CA- networks the shortest pathway between reflective rumination and the general distress variable went via brooding, and the shortest pathway between positive self-esteem and the general distress variable went via negative self-esteem. In sum, in the CA+ group six RFs had a direct shortest pathway with the general distress variable, whereas in the CA- group only three RFs had a direct shortest pathway.

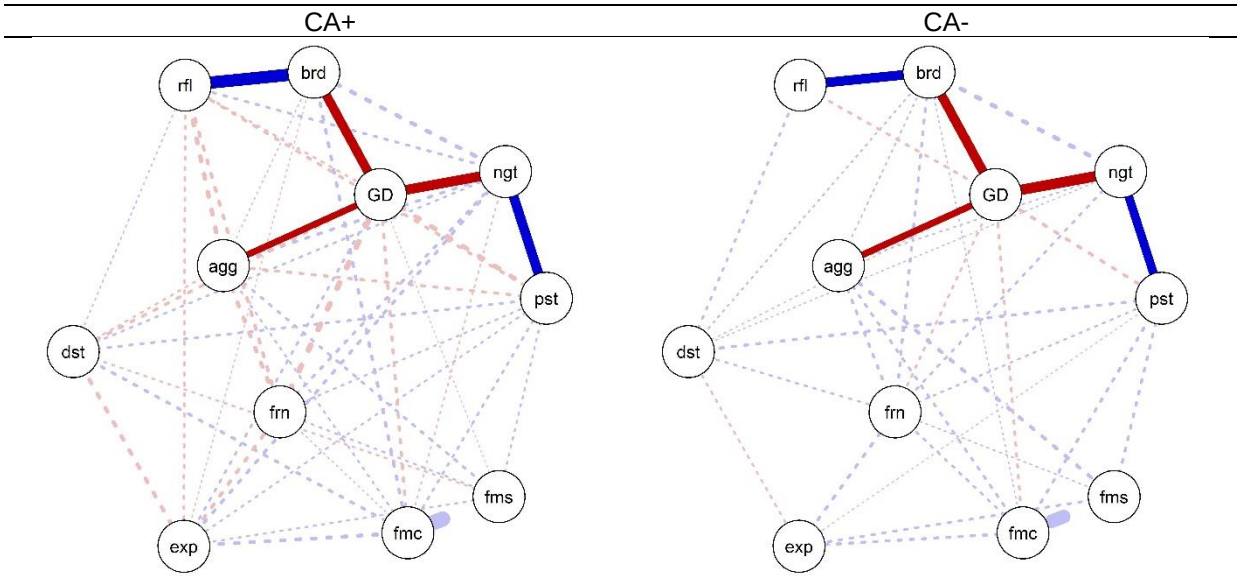



*Figure B.6.* Shortest Path Length pathways ('shortest pathways') between the RFs and the general distress variable, that are equivalent between the CA+ (*n* = 638) and the CA- (*n* = 501) group. Non-transparent, continuous lines = shortest pathway of interest. Transparent, dotted lines = all remaining regularized partial correlation connections. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family

support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress.

## APPENDIX B.14: EXPLORING THE COMPLEX INTERPLAY BETWEEN CHILDHOOD ADVERSITY, RESILIENCE FACTORS AND GENERAL DISTRESS

Interestingly, in the CA+ network seven RF-RF interrelations turned from absent to negative and three from positive to absent, upon controlling for the general distress variable. In the CA- network three RF-RF interrelations turned from positive to absent. In other words, in the CA+ network about 27 percent of the RFs are negatively interrelated and about 53 percent are positively related, upon controlling for general distress. In contrast, in the CA- network only four percent of the RF interrelations are negative and about 56 percent are positively interrelated, upon controlling for general distress. Thus, while in the CA+ network many negative related RFs may hamper each other, in the CA- network hardly any RFs seem to hamper each other. This finding was additionally supported by the degree of RF enhancement coefficient (i.e. 'general network EI', after controlling for general distress), which was significantly higher in the CA- ($EI_{CA-}$ = 2.514) than in the CA+ group ($EI_{CA+}$ = 1.790; $EI$ = 0.724, permutations = 5000, $p$ < .001). One speculative implication may be that RFs that hamper each other may alter 'RF-mental distress' relations unfavourably, resulting in an increased risk for subsequent mental health problems. However, it is important to discuss potential other, statistical explanations. We decided to control the RF-RF interrelations for general distress (see Figure B.7 panel a), to correct for potentially spurious interrelations between RFs that better can be accounted for by general distress (Elwert & Winship, 2014). However, when conditioning on general distress, the variable may contrary to our intention not have behaved as a confounder (as in Figure B.7 panel a), reducing spurious interrelations between the RFs, but may have behaved as a collider (see Figure B.7 panel b) and may have induced spurious relationships between RFs (Elwert & Winship, 2014). This may explain why in the CA+ network, seven RF interrelations that were previously absent, i.e. non-existent, became negative upon the correction for general distress. However, based on our cross-sectional data, which reveals undirected interrelations between variables (i.e. the directionality of the effect could go either way: RFs predict general distress, or vice versa), and not directed relations as in Figure B.7, we cannot with certainty draw conclusions about whether general distress behaved as expected as a confounder, or contrary to our intention as a collider.

A priori, we expected that RFs would be more strongly related to general distress in the CA+ compared to the CA- group. However, our results did not clearly show the expected pattern (see Table 3 in the main text). The zero-order correlations revealed that in the CA+ compared to the CA- group, six RFs had slightly stronger, one RF an equally strong and three RFs a slightly less strong interrelation with general distress. The regularized partial correlations revealed that in the CA+ compared to the CA- group, five RFs had slightly stronger and five RFs a slightly less strong interrelation with general distress. Moreover, the interrelation strengths of the 'RF-general distress' interrelations also seemed to be rather comparable in the CA+ and the CA- group (Pearson $R$ = .92; Spearman $R$ = .88). Therefore, we would have expected that correcting for general distress should have similar effects in both the CA+ and the CA- network. Accordingly, I believe that conditioning on a collider is unlikely to be the main explanation for why conditioning on general distress seems overall to have different effects in the CA+ and the CA- network.

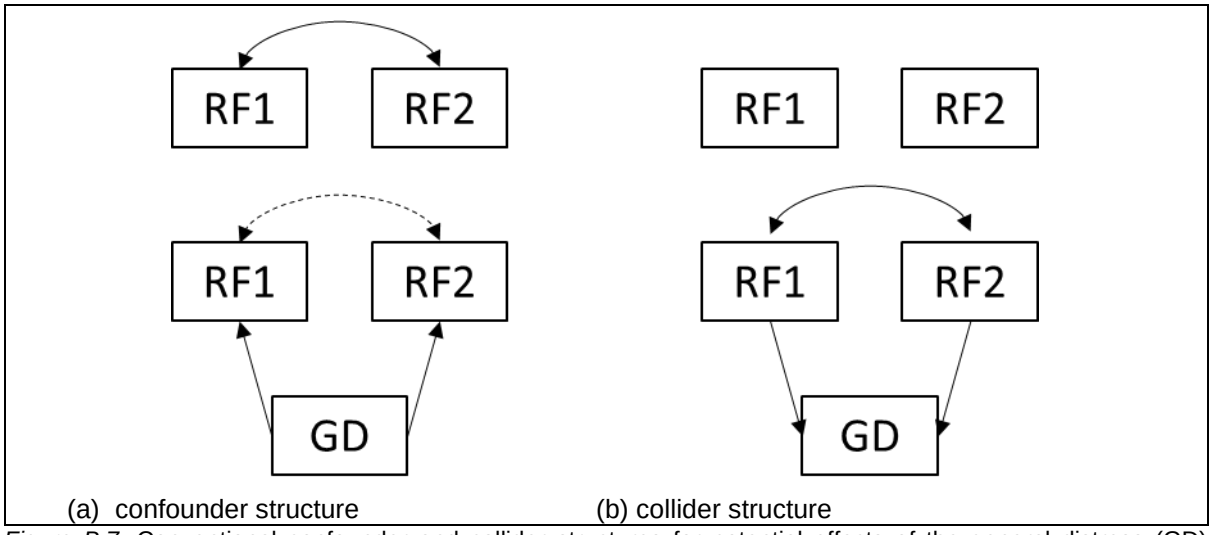



*Figure B.7.* Conventional confounder and collider structures for potential effects of the general distress (GD) variable in relation to two resilience factors (RFs), presented within directed acyclic graph (DAG) networks. The Figures are modelled along discussed example of Elwert and Winship (2014); Annual Review of Sociology; can be retrieved from https://doi.org/10.1146/annurev-soc-071913-043455.

Interestingly, even though single RFs were, in terms of interrelation strength, comparably related to distress in the two groups, we also showed that all except for one RF had significantly lower levels in the CA+ than in the CA- group and that general distress was significantly higher in the CA+ compared to the CA- group (see Table 2 in the main text). Thus in sum we found that (a) RFs are higher in the CA- group, (b) distress is higher in the CA+ group, (c) 'RF-general distress' interrelations seem to be similarly strong in the two groups, but (d) correcting for distress seems to have differing effects in the two groups. More specifically, as 'RF-general distress' interrelations seem to be similarly strong in the two groups, it is surprising that the 'RF-RF' interrelations of the two groups, which also appear to be similar, seem to be differentially impacted by the correction of general distress. We speculate that the group differences may be the result of more complex interrelations between CA, RFs and general distress, such as underlying interaction (moderation) or indirect (mediation) effects. In our pre-registered systematic review (J. Fritz, de Graaff, Caisley, van Harmelen, & Wilkinson, 2018), we defined RFs as factors that mediate and/or moderate the relationship between CA and mental distress (i.e. different types or general measure of psychopathology/distress). Thus, as we feel that we cannot disentangle with certainty whether our general distress variable in our undirected models behaved as expected as a confounder or in contrast to our expectation as a collider, but as we can investigate other statistical explanations that may help explain and understand group differences, we decided to further explore whether the RFs (as expected) moderate and/or mediate the relationship between CA and general distress, cross-sectionally.

I believe that moderation effects seem less plausible for most RFs. For a moderation effect, the relationship between the RF and general distress would have to be significantly different for the CA+ and the CA- groups, resulting in an interaction between the 'RF-general distress' slopes of the two groups (see Figure B.8b). However, as the interrelations between the RFs and general distress seemed to be similarly strong in the CA+ and the CA- group, and as the group slopes often in- or decreased in similar manners, significant interaction effects were unlikely. This conjecture was supported by our data.

For example, Figure B.8a depicts the 'RF-general distress' relationships between ruminative brooding and general distress first for the CA+ group, then for the CA- group, and in the last panel for both the CA+ and the CA- group. As can be seen, even if the CA+ group had overall higher levels of general distress at the same level of the RF, the pattern of relationship directionality (i.e. the slope) was similar for both groups. The only RF that revealed a significant interaction pattern was a low aggression potential (see Table B.7a). Yet, this finding needs to be considered with caution, as both the CA and the RF variable were dichotomous, which is suboptimal for testing interactions. Moreover, the aggression interaction seemed to behave in the opposite direction than expected. A low aggression potential reduced general distress more in the CA- than in the CA+ group (note bidirectionality). Thus, overall we conclude that moderation effects cannot explain the complex relationship between CA, RFs and general distress in our data.

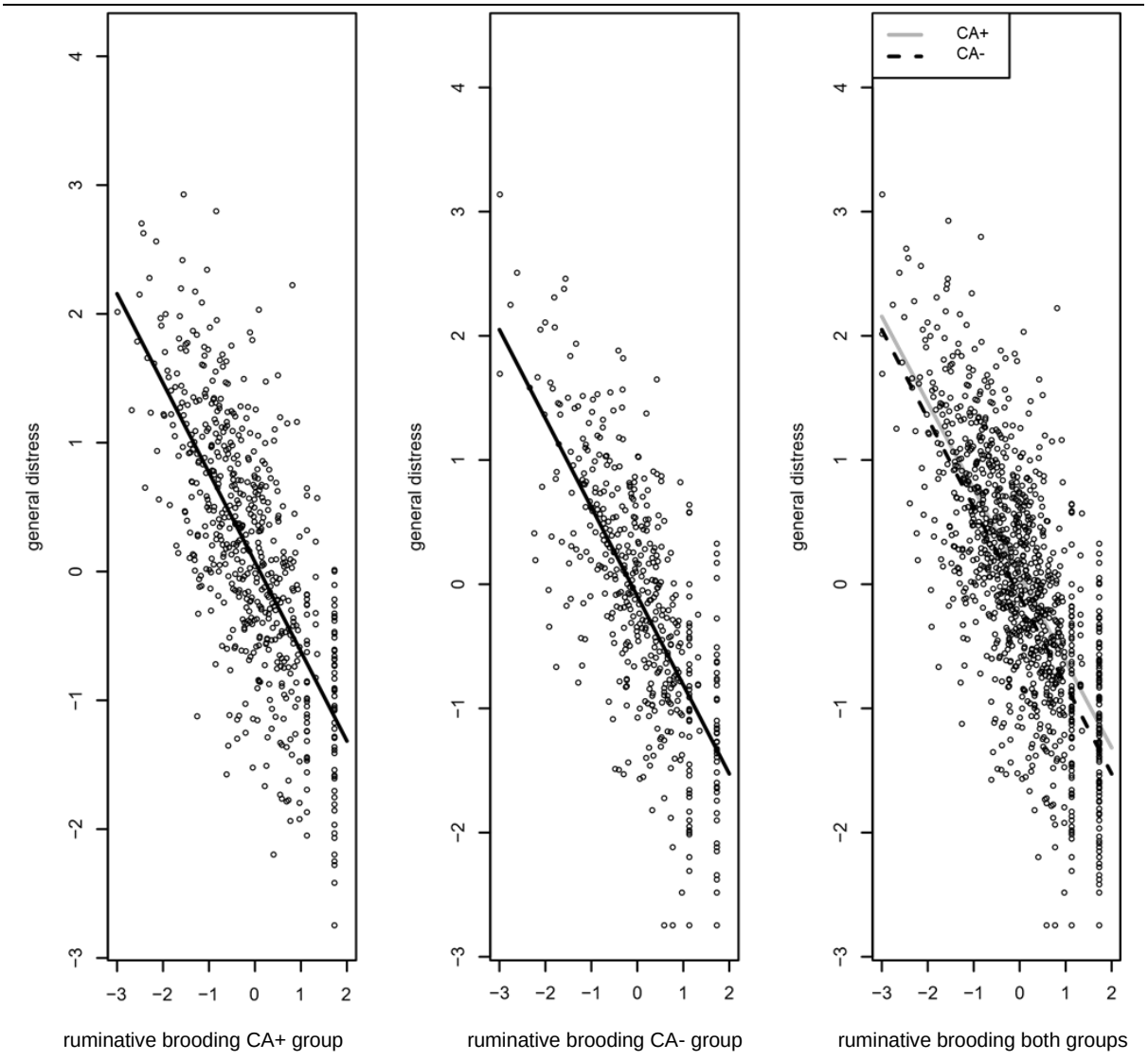
Importantly, the revealed 'RF-general distress' interrelation pattern may well indicate mediation. More specifically, CA may negatively predict the RFs and the RFs in turn may negatively predict general distress. This would mean (a) that a history of CA goes together with a higher level of general distress, (b) that a history of CA leads on average to a lower level of RFs and (c) that higher levels of the RFs in turn lead to lower levels of general distress. All three necessary prerequisites of mediation were met by our data. Moreover, for mediation to hold, the relationship between an RF and general distress can have a similar directionality pattern (i.e. slope) in the two groups, as long as either of the two groups has higher levels of the RF at the same level of general distress (see Figure B.8c and B.8d). This conjecture was clearly supported in our data. All RFs except for reflection and expressive suppression significantly mediated the relationship between CA and general distress (see Table B.7b). Yet, to verify this conjecture, longitudinal approaches are necessary, as CA should be assessed no later than the RFs and the RFs should be assessed prior to general distress. However, the cross-sectional mediation effects may to some degree explain why the correction for distress levels had differing effects on the RFs in the CA+ compared to the CA- group. Moreover, I believe that our conclusion that "CA seems to influence how resilience factors relate to each other and to current distress, potentially leading to a dysfunctional resilience factor system", was also supported by the post-hoc mediation findings, as those facilitate the idea of unfavourable 'RF-general distress' relations in the CA+ compared to the CA- group, which may increase the risk for subsequent mental health problems.

Table B.7
*RF as cross-sectional moderator and mediator for the relationship between CA and general distress*

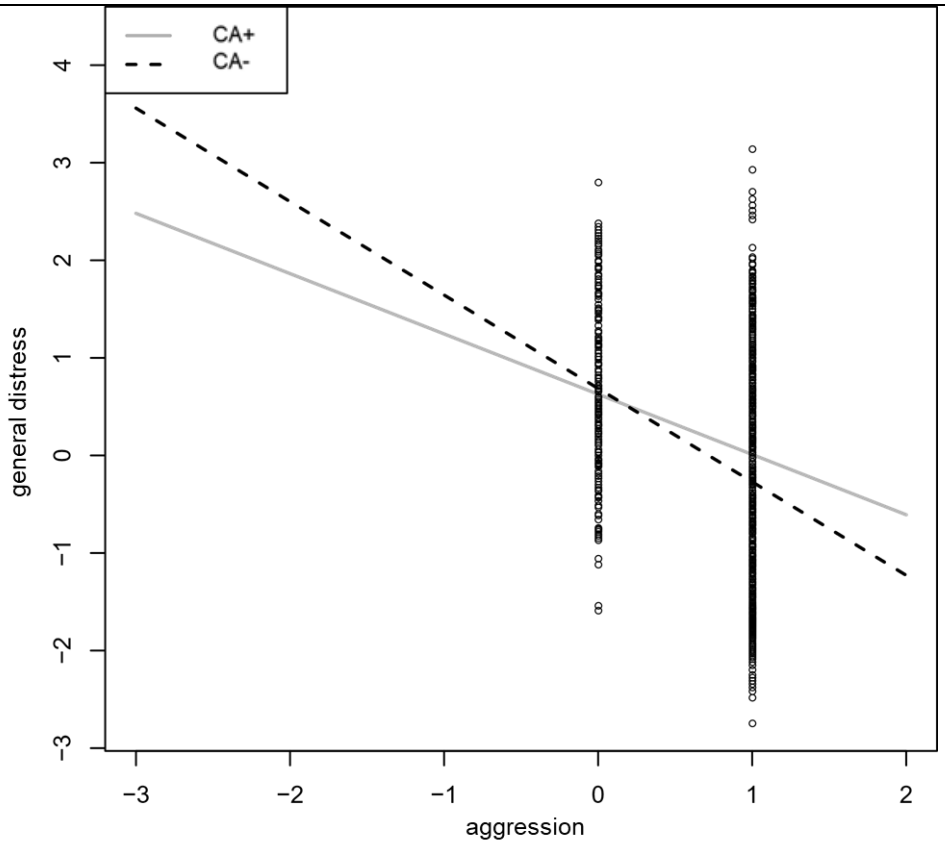
| | friend support | family support | family cohesion | positive SE | negative SE | brooding | reflection | distress tolerance | aggres-sion | exp. sup-press. |
|---|---|---|---|---|---|---|---|---|---|---|
| **B.7a: RF as cross-sectional moderator for the relationship between CA and general distress** | | | | | | | | | | |
| interaction effect est. | .02 | -.04 | -.02 | -.01 | .04 | .02 | .01 | -.09 | .34 | .10 |
| Std. error | .06 | .06 | .06 | .05 | .04 | .04 | .05 | .06 | .17 | .13 |
| *t* value | .32 | -.61 | -.32 | -.24 | .93 | .49 | .23 | -1.44 | 2.04 | .75 |
| *p* (>\|t\|) | .75 | .54 | .75 | .81 | .36 | .62 | .82 | .15 | **.04*** | .45 |
| Adjusted analysis $R^2$ | .15 | .11 | .17 | .32 | .56 | .51 | .22 | .09 | .09 | .02 |
| **B.7b: RF as cross-sectional mediator for the relationship between CA and general distress** | | | | | | | | | | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| indirect effect est. | .05 | .05 | .15 | .17 | .17 | .13 | .03 | .07 | .06 | -.001 |
| Std. error | .02 | .02 | .03 | .03 | .04 | .04 | .03 | .02 | .02 | .01 |
| *z* value | 2.25 | 2.75 | 5.75 | 5.00 | 3.85 | 2.97 | 1.23 | 3.93 | 3.61 | -.22 |
| *p* value | **.03*** | **.01**** | **<.001***** | **<.001***** | **<.001***** | **.003**** | .22 | **<.001***** | **<.001***** | .82 |
| 95% *CI* | .01-.09 | .02-.09 | .10-.21 | .10-.23 | .08-.26 | .04-.21 | -.02-.09 | .03-.10 | .03-.09 | -.01-.01 |
| general distress $R^2$ | .15 | .11 | .17 | .32 | .56 | .52 | .22 | .09 | .09 | .02 |

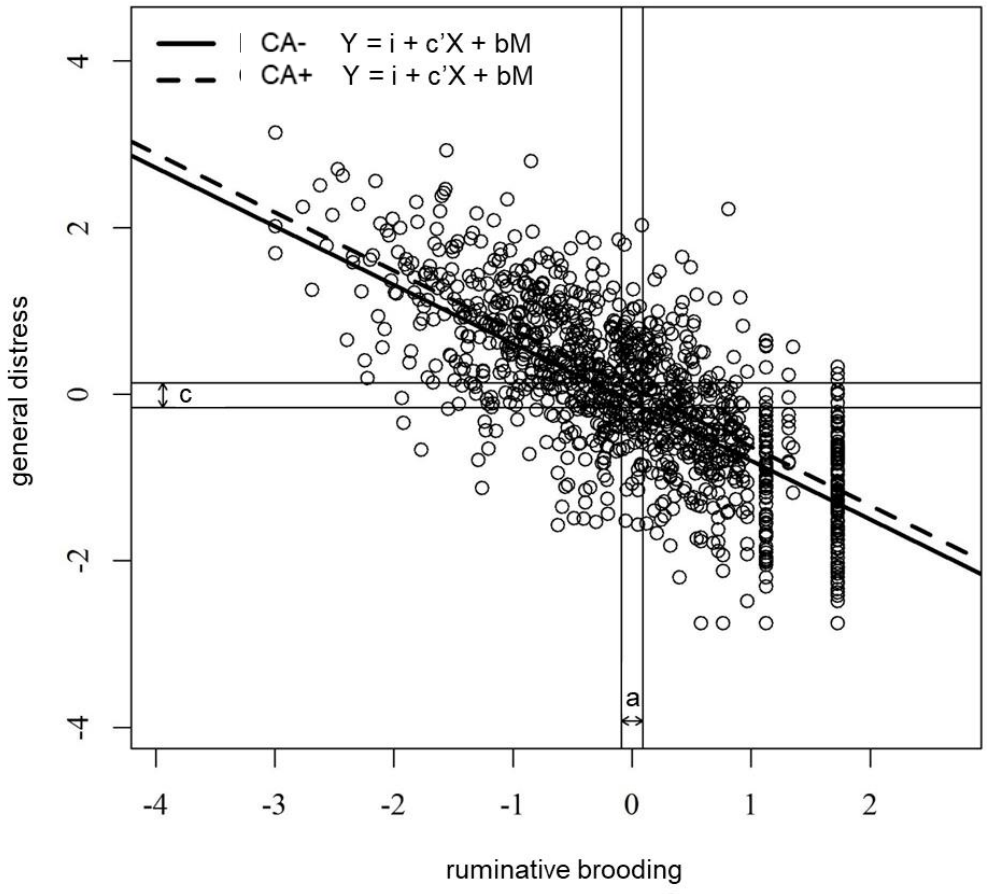
Note. SE = self-esteem; Exp. suppress. = expressive suppression; Est. = estimate; Std. error = Standard error; CI = confidence interval.



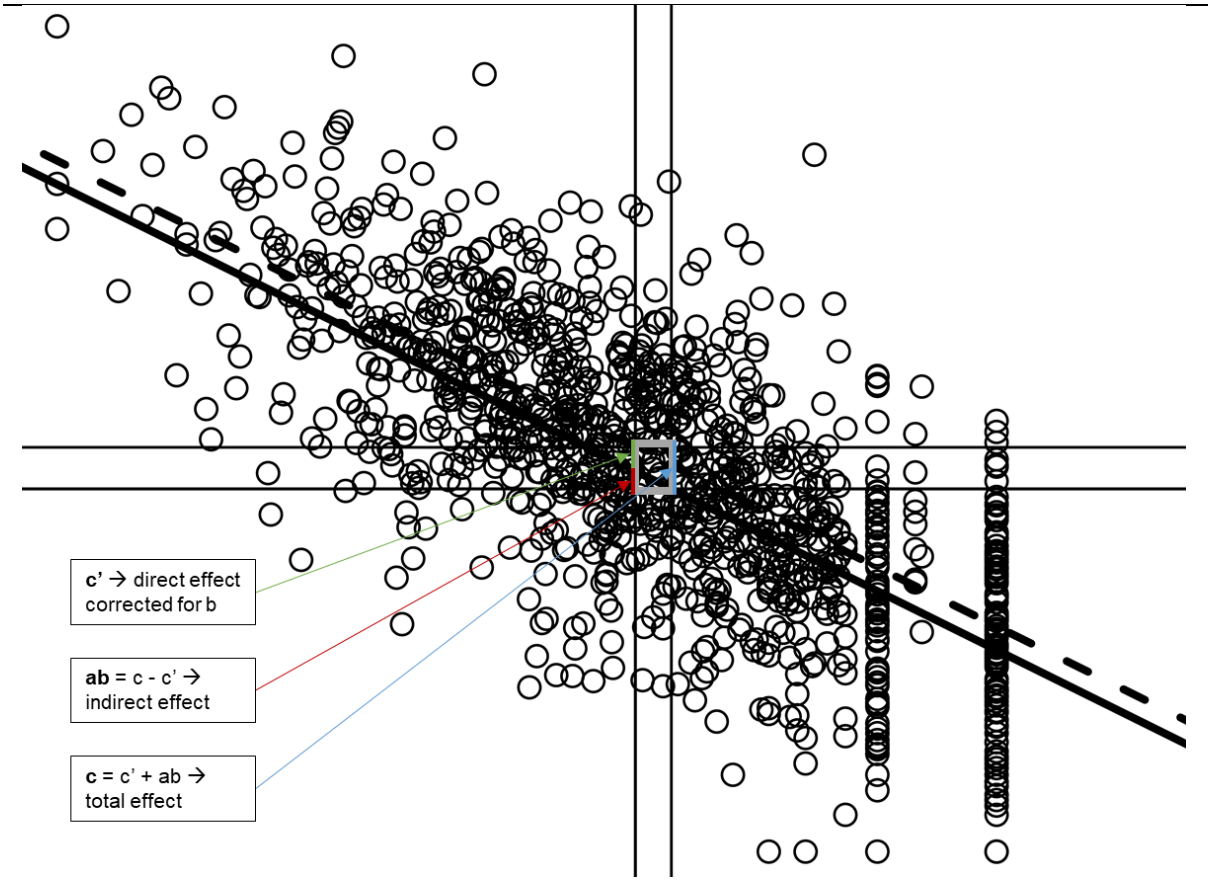

(a) Moderation: no significant interaction effect




(b) Moderation: significant interaction effect




(c) Mediation: significant indirect effect




(d) Mediation: stipulation of the direct, indirect and total effects

*Figure B.8.* Moderation and mediation example for RFs as moderator and mediator for the relationship between CA and general distress. Panel (a) depicts the RF low brooding, which has no significant interaction effect. Panel (b) depicts the RF low aggression potential, which has a significant interaction effect. Panel (c) depicts the RF low brooding, which has a significant indirect effect. Panel (d) stipulation of the direct, indirect and total effect of the mediation analysis for low brooding. The mediation figures are modelled using an adapted script from M. S. Fritz and MacKinnon (2008a); Behavior Research Methods: https://doi.org/10.3758/BRM.40.1.55; the original scripts can be found in the belonging supplement material (M. S. Fritz & MacKinnon, 2008b) at: https://link.springer.com/article/10.3758/BRM.40.1.55#SupplementaryMaterial.

# APPENDIX C
# Appendix Chapter 4

## APPENDIX C.1: RATIONALE FOR CHANGES IN VARIABLES SINCE CHAPTER 3

In Chapter 3 (or J. Fritz, Fried, Goodyer, Wilkinson, & van Harmelen, 2018) we used a distress index computed by Brodbeck, Abbott, Goodyer, and Croudace (2011). This index was however only computed for age 14. As we here wanted to compare two occasions, we decided to compute a separate general distress factor including information for both occasions, so that we could apply invariance constraints over the two time points. This was for example necessary to compare the latent means. Moreover, in comparison to Brodbeck and colleagues (2011) we simplified the index (e.g. through only using the short form rather than the complete version of the used depression scale), to ensure feasibility of the computation of the invariance models (particularly because of the increased computation time with regard to the multiple imputed data sets).

As the rumination questionnaire underwent revision between our two occasions, different versions of the questionnaire were used at the two occasions. The ruminative reflection factor stayed the same for both time points, however, we had to use another ruminative brooding factor than in Chapter 3 (or J. Fritz, Fried, et al., 2018) as only two of the five originally used items were available for both occasions. Here I report results based on a ruminative brooding approach that has been established by Burwell and Shirk (2007; 6 items) for a version of the rumination questionnaire that matches the one used at our second occasion, as five of the six identified items of that approach were available for both of our occasions. We also analysed the network models excluding the brooding variable, which revealed similar results, see Appendix C.2.

## APPENDIX C.2: NETWORK MODELS THIS TIME EXCLUDING THE BROODING VARIABLE

The following three figures depict CA+ and CA- networks as presented in Chapter 4, however this time excluding the brooding variable. The models were estimated separately for age 14 and 17, as well as (1) once without the general distress variable, (2) once with the general distress variable, and (3) once corrected for the general distress variable. At age 14, the network invariance test was not significant for the networks without the general distress variable (M = .14, p = .41; see Figure C.1), but the global network expected influence differed between the CA+ and the CA- RF networks ($EI_{CA+}$ = 2.59, $EI_{CA-}$ = 2.96, EI = 0.37, p = .02). More specifically, the CA+ network was less positively connected than the CA- network. Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .12, p = .64; $EI_{CA+}$ = 2.93, $EI_{CA+}$ = 2.79, EI = 0.14, p = .31). When we compared the RF networks for age 14 and age 17, the two CA+ network were not invariant over time, in other words, they did vary over time (M = .23, p = .002). Moreover, the RFs in the age 14 network were less positively interrelated than in the age 17 network ($EI_{14}$ = 2.59, $EI_{17}$ = 2.93, EI = 0.34, p = .003). The age 14 and age 17 CA- networks did however not differ with regard to their global network structure (M = .15, p = .45; $EI_{14}$ = 2.96, $EI_{17}$ = 2.79, EI = 0.17, p = .26).

For the networks with the general distress variable, the network invariance test (M = .19, p = .12; see Figure C.2) was not significant at age 14. However, the global network expected influence differed significantly between the CA+ and the CA- networks ($EI_{CA+}$ = -0.19, $EI_{CA-}$ = 0.75, EI = 0.94, p < .01). Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .15, p = .51; $EI_{CA+}$ = 0.13, $EI_{CA-}$ = 0.55, EI = 0.42, p = .19). When we compared the networks for age 14 and age 17, we did not find any significant global network structure differences; neither for adolescents with (M = .15, p = .19; $EI_{14}$ = -0.19, $EI_{17}$ = 0.13, EI = 0.32, p = .35) nor for adolescents without a history of adversity (M = .14, p = .62; $EI_{14}$ = 0.75, $EI_{17}$ = 0.55, EI = 0.20, p = .50).

For the networks corrected for the general distress variable, the network invariance test for the CA+ and the CA- group (M = .19, p = .10; see Figure C.3) was not (or only very marginally) significant, at age 14. In contrast, the global network expected influence differed between the CA+ and the CA- networks ($EI_{CA+}$ = 1.36, $EI_{CA-}$ = 2.09, EI = 0.73, p = .001). Those findings were again only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .12, p = .82; $EI_{CA+}$ = 1.69, $EI_{CA-}$ = 1.92, EI = 0.24, p = .27). When we compared the networks for age 14 and age 17, we once more did not find any significant global network structure differences; neither for adolescents with (M = .12, p = .53; $EI_{14}$ = 1.36, $EI_{17}$ = 1.69, EI = 0.33, p = .17) nor for adolescents without a history of adversity (M = .14, p = .58; $EI_{14}$ = 2.09, $EI_{17}$ = 1.92, EI = 0.17, p = .41).

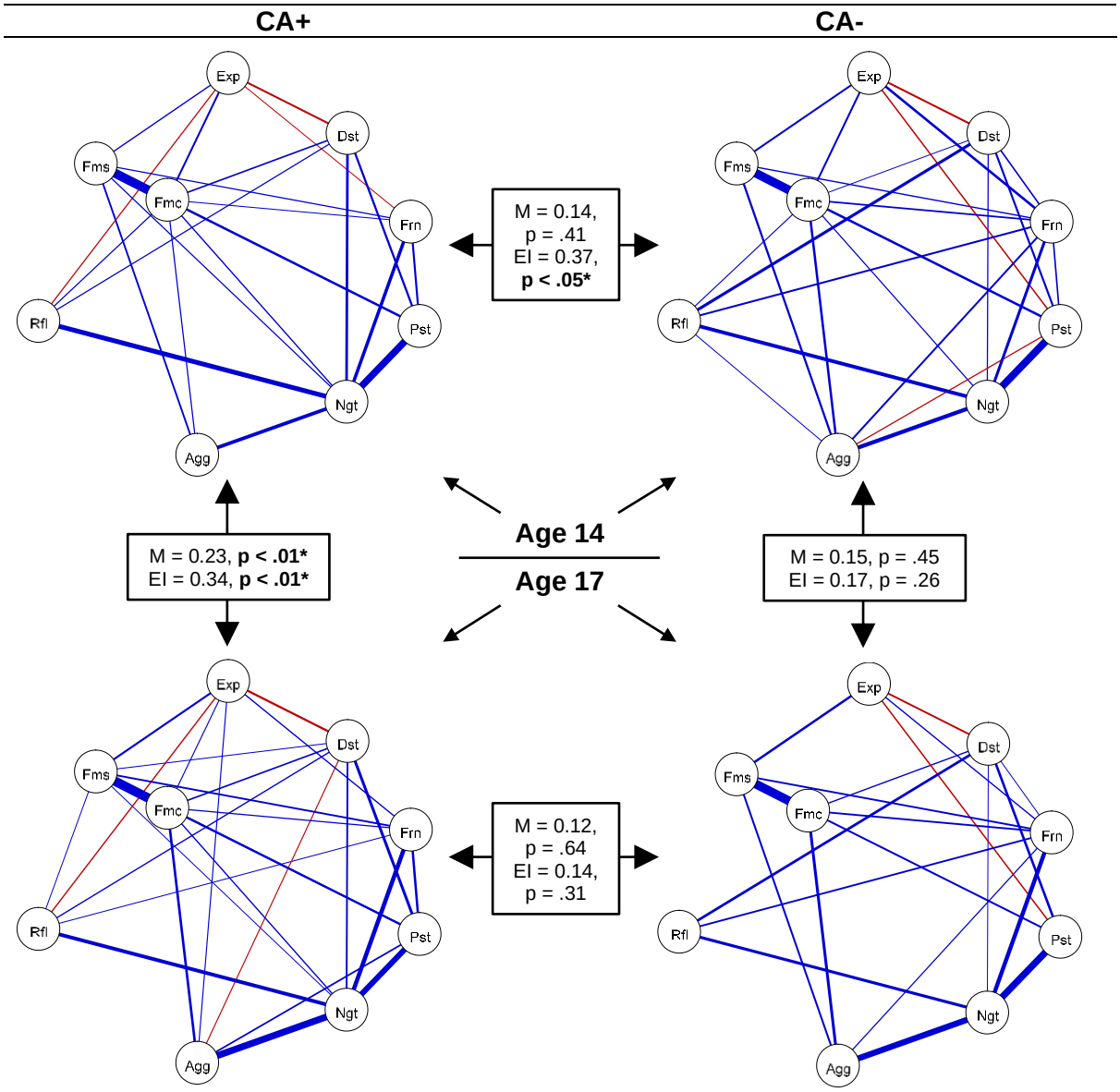



*Figure C.1.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for age 14 (upper panel) and age 17 (lower panel) without the brooding and the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

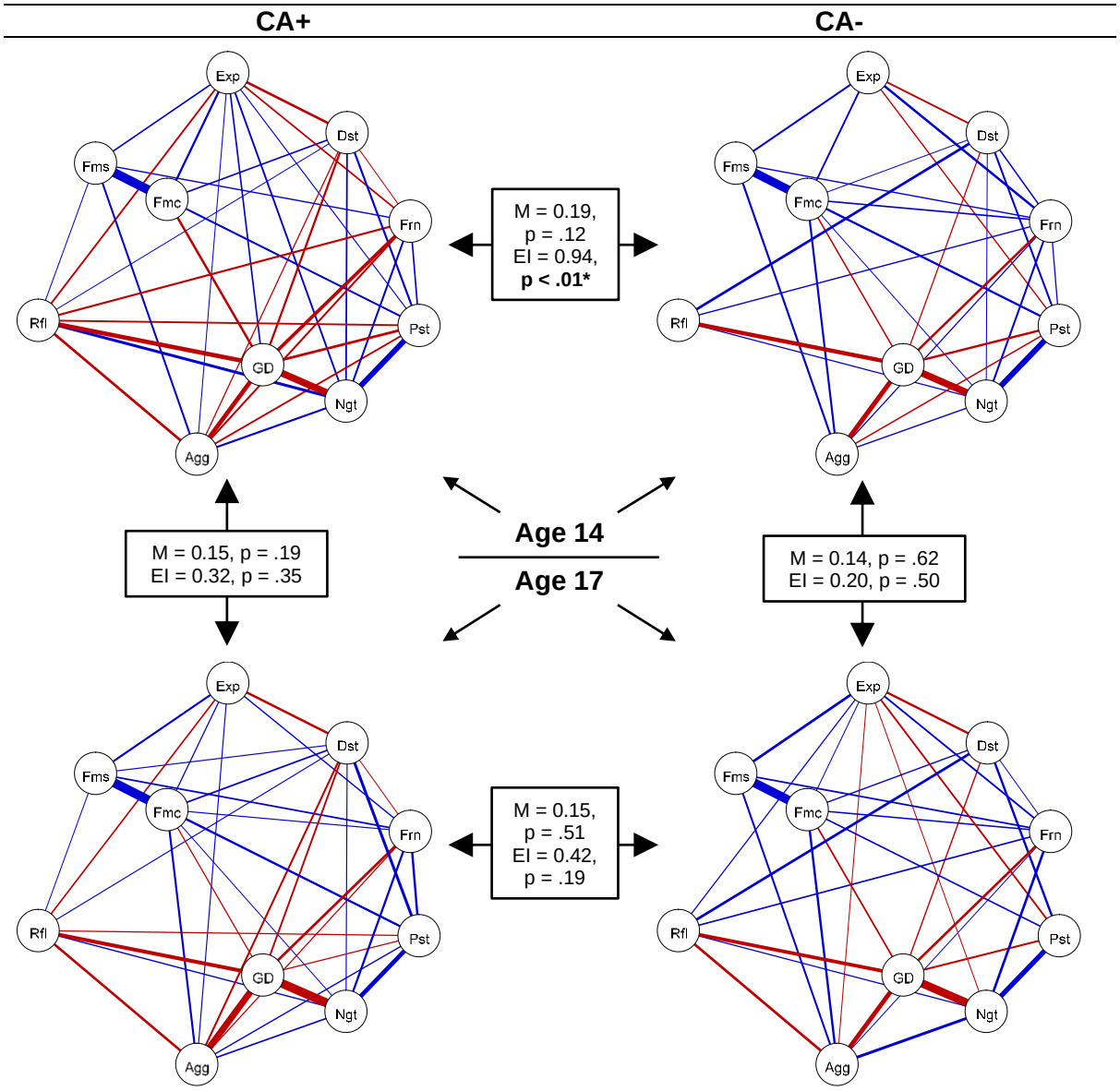



*Figure C.2.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for age 14 (upper panel) and age 17 (lower panel) without the brooding variable, but with the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

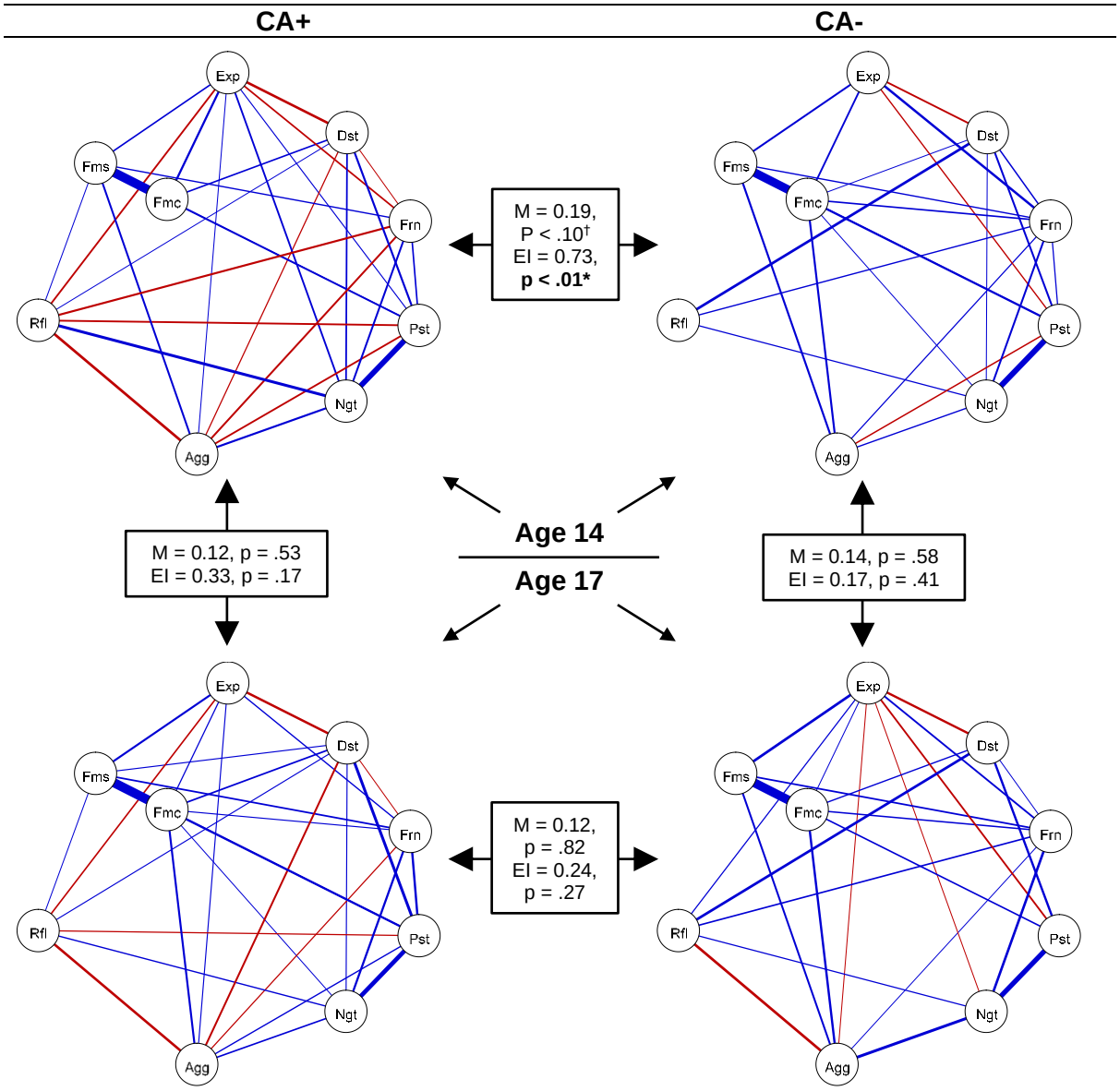



*Figure C.3.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for age 14 (upper panel) and age 17 (lower panel) without the brooding variable and corrected for the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

## APPENDIX C.3: OVERVIEW OF USED R PACKAGES, INCLUDING THEIR VERSION NUMBER AND REFERENCE

We conducted all analyses in R version 3.5.1, and used the below packages (see Table C.1), and further dependencies these packages load.

Table C.1
*Used R packages, including their version number and reference*

| **Package (version number)** | **Reference** |
|---|---|
| mice (3.5.0) | (van Buuren & Groothuis-Oudshoorn, 2011) |
| dplyr (0.7.7) | (Wickham, François, Henry, & Müller, 2018) |
| pastecs (1.3.21**)** | (Grosjean & Ibanez, 2018) |
| coin (1.2-2) | (Hothorn, Hornik, van de Wiel, & Zeileis, 2008) |
| reshape (0.8.8) | (Wickham, 2007) |
| sjPlot (2.6.2) | (Lüdecke, 2018) |
| lavaan (0.6-4) | (Rosseel, 2012) |
| semTools (0.5-1.933) | (Jorgensen, Pornprasertmanit, Schoemann, & Rosseel, 2018) |
| ggplot2 (3.1.0) | (Wickham, 2016) |
| qgraph (1.5) | (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012) |
| bootnet (1.1.0) | (Epskamp, Borsboom, & Fried, 2018) |
| NetworkComparisonTest (2.0.1) | (van Borkulo, 2018) |

## APPENDIX C.4: MISSING DATA PATTERNS AND MISSINGNESS PREDICTORS

We evaluated differences between those participants who had full data and participants who had missingness, either due to attrition or incidental missingness, as stipulated in Table C.2. All RFs and the general distress variable had a small number of participants with entire missingness at both time points (50 to 78 participants). A minor subset of participants did not provide data at occasion1, but at occasion 2 for some of the scales (10 to 30 participants per scale). There was more attrition for people who provided data at occasion 1 but not at occasion 2 (123 to 294 per scale). There was a range of participants with incidentally missing items, which differed largely per scale (from 4 for aggression to 152 for general distress).

We also investigated whether the missingness was predictable (see Table C.3). We identified that missingness on all RFs and general distress could be explained by CA. Moreover, for seven RFs and distress missingness was also explained by being male and by having a low mood (MFQ levels). A psychiatric history prior to the age of 14 explained additionally missingness in six RFs and general distress. Overall, we excluded 50 participants who had more than 85% of missing items across the scales, which resulted in 1188 remaining participants. Among those 1188 all had less than 59% missing items. On average the items had 9% missingness.

Table C.2
*Missingness patterns (N= 1238)*

| Variable | No data | Full data | Missing T2 | Missing T1 | Incidental M |
|---|---|---|---|---|---|
| FRN | 051 | 941 | 192 | 030 | 024 |
| FMS | 054 | 915 | 190 | 028 | 051 |
| FMC | 054 | 917 | 190 | 028 | 049 |
| PST | 050 | 955 | 180 | 027 | 026 |
| NGT | 050 | 961 | 180 | 027 | 020 |
| BRD | 050 | 1004 | 123 | 029 | 032 |
| RFL | 050 | 1000 | 123 | 030 | 035 |
| DST | 073 | 849 | 294 | 010 | 012 |
| AGG | 050 | 975 | 180 | 029 | 004 |
| EXP | 078 | 854 | 292 | 014 | 000 |
| GD | 050 | 830 | 179 | 027 | 152 |

*Note.* Incidental M. = incidental missingness, FRN = friendship support, FMS = family support, FMC = family cohesion, PST = positive self-esteem, NGT = negative self-esteem, BRD = brooding, RFL = reflection, DST = distress tolerance, AGG = aggression, EXP = expressive suppression, GD = general distress.

Table C.3
*Missingness predictors*

| | | U/ $\chi^2$ (df) | *p* | Cross tabs (1 = no missing; 2 = missing) |
|---|---|---|---|---|
| GD | CA | 7.0557 (1) | <0.01** | 1 2<br>CA- 374 127<br>CA+ 429 209 |
| | Gender | 5.9034 (1) | <0.05* | 1 2 |

| | | | | |
|---|---|---|---|---|
| | | | | female 458 216<br>male 345 219 |
| | MFQ | 123280 | <0.001*** | 1 M: 15.3300<br>2 M: 17.8711485 |
| | Age-14 prior psychiatric history (PP; yes = PP+; no = PP-) | 5.6529 (1) | <0.05* | 1 2<br>PP- 623 286<br>PP+ 180 116 |
| FRN | CA | 11.361 (1) | <0.001*** | 1 2<br>CA- 423 78<br>CA+ 486 152 |
| | Gender | 10.95 (1) | <0.001*** | 1 2<br>female 521 153<br>male 388 176 |
| | MFQ | 103140 | <0.05* | 1 M = 15.7665<br>2 M = 17.3571429 |
| | Age-14 PP | 3.9534 (1) | <0.05* | 1 2<br>PP- 699 210<br>PP+ 210 86 |
| FMS | CA | 20.05 (1) | <0.001*** | 1 2<br>CA- 421 80<br>CA+ 464 174 |
| | Gender | 3.9295 (1) | <0.05* | 1 2<br>female 498 176<br>male 387 177 |
| | MFQ | 109500 | <0.001*** | 1 M: 15.5386<br>2 M: 17.8088737 |
| | Age-14 PP | 5.8008 (1) | <0.05* | 1 2<br>PP- 684 225<br>PP+ 201 95 |
| FMC | CA | 19.544 (1) | <0.001*** | 1 2<br>CA- 421 80<br>CA+ 465 173 |
| | Gender | 4.7008 (1) | <0.05* | 1 2<br>female 500 174<br>male 386 178 |
| | MFQ | 109270 | <0.001*** | 1 M: 15.5271<br>2 M: 17.843003 |
| | Age-14 PP | 5.9935 (1) | <0.05* | 1 2<br>PP- 685 224<br>PP+ 201 95 |
| PST | CA | 7.9443 (1) | <0.01** | 1 2<br>CA- 425 76<br>CA+ 498 140 |
| | Gender | 12.51 (1) | <0.001*** | 1 2<br>female 530 144<br>male 393 171 |
| | MFQ | 102010 | 0.094 | 1 M: 15.8959<br>2 M: 16.9495798 |
| | Age-14 PP | 3.736 (1) | 0.053 | 1 2<br>PP- 709 200<br>PP+ 214 82 |
| NGT | CA | 10.722 (1) | <0.01** | 1 2<br>CA- 430 71<br>CA+ 498 140 |
| | Gender | 10.222(1) | <0.01** | 1 2<br>female 530 144<br>male 398 166 |
| | MFQ | 99465 | 0.062 | 1 M: 15.8263<br>2 M: 17.2489270 |
| | Age-14 PP | 1.8092 (1) | 0.18 | 1 2<br>PP- 709 200<br>PP+ 219 77 |
| AGG | CA | 11.568 (1) | <0.001*** | 1 2<br>CA- 436 65<br>CA+ 505 133 |
| | Gender | 18.327 (1) | <0.001*** | 1 2 |

| | | | | |
|---|---|---|---|---|
| | | | | female 562 112<br>male 413 151 |
| | MFQ | 84244 | 0.13 | 1 M: 15.9066<br>2 M: 17.1881720 |
| | Age-14 PP | 2.3499 (1) | 0.13 | 1 2<br>PP- 745 164<br>PP+ 230 66 |
| BRD | CA | 9.5907 (1) | <0.01** | 1 2<br>CA- 446 55<br>CA+ 525 113 |
| | Gender | 4.2525 (1) | <0.05* | 1 2<br>female 544 130<br>male 427 137 |
| | MFQ | 83354 | <0.05* | 1 M: 15.8412<br>2 M: 17.4947368 |
| | Age-14 PP | 1.4102 (1) | 0.24 | 1 2<br>PP- 740 169<br>PP+ 231 65 |
| RFL | CA | 7.2547 (1) | <0.01** | 1 2<br>CA- 442 59<br>CA+ 525 113 |
| | Gender | 3.7544 (1) | 0.053 | 1 2<br>female 541 133<br>male 426 138 |
| | MFQ | 83594 | <0.05* | 1 M: 15.8002<br>2 M: 17.6649485 |
| | Age-14 PP | 1.8251 (1) | 0.18 | 1 2<br>PP- 738 171<br>PP+ 229 67 |
| DST | CA | 13.384 (1) | <0.001*** | 1 2<br>CA- 399 102<br>CA+ 446 192 |
| | Gender | 0.29893 (1) | 0.59 | 1 2<br>female 465 209<br>male 380 184 |
| | MFQ | 112880 | <0.001*** | 1 M: 15.2343<br>2 M: 18.3012048 |
| | Age-14 PP | 8.6094 (1) | <0.01** | 1 2<br>PP- 658 251<br>PP+ 187 109 |
| EXP | CA | 20.024 (1) | <0.001*** | 1 2<br>CA- 407 94<br>CA+ 443 195 |
| | Gender | 0.47049 (1) | 0.49 | 1 2<br>female 471 203<br>male 383 181 |
| | MFQ | 111640 | <0.001*** | 1 M: 15.2452<br>2 M: 18.3487654 |
| | Age-14 PP | 9.8232 (1) | <0.01** | 1 2<br>PP- 666 243<br>PP+ 188 108 |

*Note.* CA = childhood adversity, MFQ = Mood and Feelings Questionnaire, FRN = friendship support, FMS = family support, FMC = family cohesion, PST = positive self-esteem, NGT = negative self-esteem, BRD = brooding, RFL = reflection, DST = distress tolerance, AGG = aggression, EXP = expressive suppression, GD = general distress.

## APPENDIX C.5

### PART A: RATIONALE FOR USING FACTOR SCORES, INSTEAD OF SUM SCORES

We decided to use factor scores, instead of sum scores, for two reasons. Firstly, to remove as much measurement error as possible from the latent resilience factor (RF) variables. In most published network analysis manuscripts author have used item level data. Yet, here we were not interested in the individual items but in RF constructs which were derived from a previous systematic review. As all RFs (except for expressive suppression) consisted of more than 3 items we could apply factor analyses to effectively reduce measurement error. A similar method would have been to use latent network modelling, which does the same but estimates the factor scores and the network models in one step (Epskamp et al., 2017). Upon closer inspection we concluded that latent network modelling is as yet only (or at least particularly) applicable to smaller models. The second reason for using factor scores was that when using sum scores one assumes that all items have the same importance and hence go with the same weight into the construct (i.e. tau equivalence). However, when using factor scores, the factor loadings enable every item to have a unique weight for the latent construct, which means that items can differ in importance, enhancing construct validity. We felt that this point was particularly important as many of our used (sub-)scales did not consist of a large number of items (with exception for the general distress factor).

For completeness, we additionally performed our analyses based on sum scores. I added the results for mean change analyses with sum scores to Appendix C.6 Part B and the results for network analyses with sum scores to Appendix C.12. The sum score results were overall similar to the results for fully invariant factor scores.

### PART B: MODEL SPECIFICATIONS AND MODEL FIT FOR THE THREE ESTIMATED INVARIANCE LEVELS OF THE CATEGORICAL LONGITUDINAL CONFIRMATORY FACTOR ANALYSES FOR THE RESILIENCE FACTORS AND THE DISTRESS INDEX, AS WELL AS BOX-AND-WHISKER PLOTS WITH INDIVIDUAL DATA POINTS FOR THE RESULTING FACTOR SCORES

As we aimed to compare two time points, we estimated longitudinal CFAs (LCFAs) separately for each RF and the general distress variable. Given that all of the RF items (as well as the general distress items) were assessed with three to six answer categories, we computed categorical LCFAs and treated the items as ordinal (i.e. ordered categorical) indicators (Liu et al., 2017; Muthen & Asparouhov, 2002; Sass, 2011; Wu & Estabrook, 2016). Accordingly, we used the weighted least squares mean and variance adjusted (WLSMV) estimator. The categorical LCFAs were specified as shown in Figure C.4 (which is modelled along examples of Liu et al., 2017). We identified the model as suggested by Wu and Estabrook (2016), using the theta parametrization. We estimated, a configural (i.e. baseline) model, a strong invariance and a full invariance model. For the strong invariance LCFAs we equated item loadings and item thresholds across the two time points (i.e. age 14 and 17), fixed all item intercepts to 0, the item variance of the first time point to 1, the latent factor mean of the first time point to 0, and the latent factor variance of the first time point to 1 (item covariances and the latent factor covariance were freely estimated). For the full invariance LCFAs we again equated item loadings and item thresholds

across the two time points (i.e. age 14 and 17) and fixed all item intercepts to 0, this time however we fixed all item variances to 1, both latent factor means to 0, and both latent factor variances to 1 (item covariances and the latent factor covariance were again freely estimated). A model specification overview can be found in Table C.4. Table C.5 depicts the fit indices for all models. We only applied modification indices when they were theoretically justified. We intended to pool over the fit indices of the 10 result sets (i.e. one for each imputation data set). We however discovered that for many of our models the pooling of fit indices resulted in either a negative or a close to negative chi-square statistics. When the chi-square statistic is negative, it needs to be set to zero, as the pooled fit otherwise cannot be computed. Unfortunately, a zero chi-square results in an arbitrary model fit. Therefore, we decided to report the fit indices of the separate models, which we consider more informative in this case (see Table C.5). We do additionally provide the pooled standardized root mean residual (pooled SRMR) as this fit measure does not rely on the chi-square statistic (i.e. it represents the standardized difference between the observed and the predicted correlation) and is therefore reliable for our models. All models seemed to fit acceptably. Factor scores derived from the aggression models were however so poorly distributed that we had to binarize those scores. Distribution plots (i.e. box-and-whisker plots with individual data points) for the RFs (except for expressive suppression and aggression) and the general distress variable are depicted in Figure C.5.

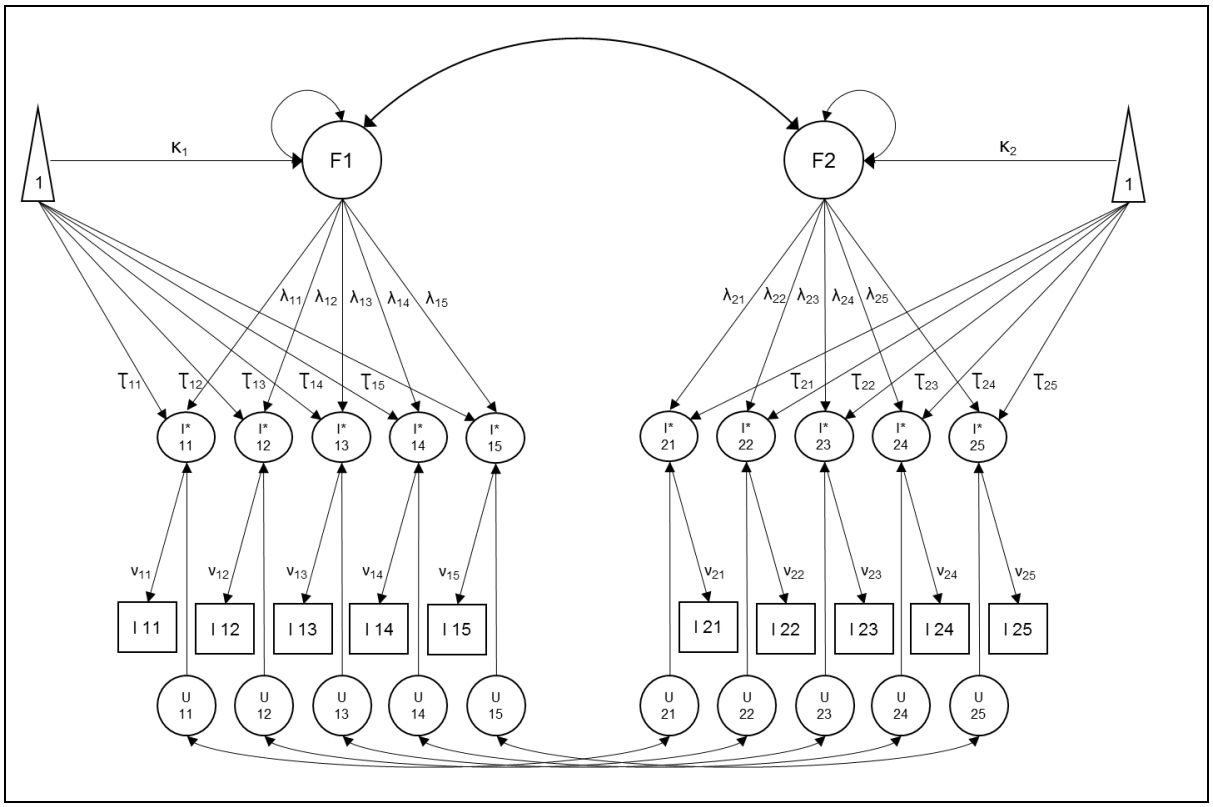



*Figure C.4*. Longitudinal, categorical CFA model, with 5 categorical items assessed at two time points. The model is defined as follows: F = **common latent factor** (with the factor number indicating the corresponding time point); κ = **common latent factor means** (with the subscript number indicating the corresponding latent factor); I = **categorical observed items**; I* = **continuous latent item responses** inferred from the categorical observed items and described by the item thresholds; λ = **item loadings**; τ= **item intercepts**; ν = **item thresholds** (the number of thresholds is not depicted as every ordinal item has multiple thresholds, namely one less than the number of measured categories); U = **unique latent (item) factors;** for indicators with two numbers, the first number refers to the time point and the second to the item number; ↔ = two-sided arrows

indicate (auto-co)variances. The Figure is modelled along examples of Liu, Millsap, West, Tein, Tanaka and Grimm (2017): https://doi.org/10.1037/met0000075.

Table C.4
*Model specifications for the three estimated invariance levels of the categorical LCFAs*

**(1) Configural Model**

*Estimated parameters:*

1. λ = **factor loadings:** all freely estimated
2. ν = **items thresholds:** all freely estimated
3. auto-covar(U) = **unique latent (item) factor auto-covariances:** between the corresponding time 1 and time 2 unique latent (item) factors are all freely estimated
4. auto-covar(F) = **common latent factor auto-covariance:** between the corresponding time 1 and time 2 common latent factor is freely estimated

*Parameters fixed for both time points:*

1. τ = **item intercepts:** all fixed to zero
2. var(U) = **unique latent (item) factor variances**: are all fixed to one
3. κ = **common latent factor means:** all fixed to zero
4. var(F) = **common latent factors variances**: are all fixed to one

*Parameters fixed for only the first but estimated for the second time point:*

-

*Equated parameters across time:*

-

**(2) Strong Invariance Model**

*Estimated parameters:*

1. auto-covar(U) = **unique latent (item) factor auto-covariances:** between the corresponding time 1 and time 2 unique latent (item) factors are all freely estimated
2. auto-covar(F) = **common latent factor auto-covariance:** between the corresponding time 1 and time 2 common latent factor is freely estimated

*Parameters fixed for both time points:*

1. τ = **item intercepts:** all fixed to zero

*Parameters fixed for only the first but estimated for the second time point:*

1. var(U) = **unique latent (item) factor variances:** fixed to one only for the first, but not the second time point
2. κ = **common latent factor means:** fixed to zero only for the first, but not the second time point
3. var(F) = **common latent factor variances:** fixed to one only for the first, but not the second time point

*Equated parameters across time:*

1. λ = **factor loadings:** all equated across time
2. ν = **items thresholds:** all equated across time

**(3) Full Invariance Model**

*Estimated parameters:*

1. auto-covar(U) = **unique latent (item) factor auto-covariances:** between the corresponding time 1 and time 2 unique latent (item) factors are all freely estimated

2. auto-covar(F) = **common latent factor auto-covariance:** between the corresponding time 1 and time 2 common latent factor is freely estimated

*Parameters fixed for both time points:*

1. τ = **item intercepts:** all fixed to zero
2. var(U) = **unique latent (item) factor variances:** all fixed to one
3. κ = **common latent factor means:** all fixed to zero
4. var(F) = **common latent factor variances:** all fixed to one

*Parameters fixed for only the first but estimated for the second time points:*

-

*Equated parameters across time:*

1. λ = **factor loadings:** all equated across time
2. v = **items thresholds:** all equated across time

Table C.5
*Longitudinal, categorical confirmatory factor analyses conducted with the WLSMV estimator*

| Model | CFI | TLI | RMSEA | RMSEA 90% CI | RMSEA 90% CI | $Chi^2$ |
|---|---|---|---|---|---|---|
| *Friendship support (Goodyer et al., 1989), 5 items, 0 unique item covariances* | | | | | | |
| **CM-1** | 0.976 | 0.962 | 0.079 | 0.070 | 0.089 | 244.996 |
| **CM-2** | 0.975 | 0.961 | 0.086 | 0.077 | 0.096 | 285.431 |
| **CM-3** | 0.982 | 0.973 | 0.083 | 0.074 | 0.092 | 264.909 |
| **CM-4** | 0.974 | 0.960 | 0.086 | 0.077 | 0.095 | 283.386 |
| **CM-5** | 0.978 | 0.965 | 0.085 | 0.076 | 0.094 | 275.672 |
| **CM-6** | 0.974 | 0.959 | 0.083 | 0.074 | 0.093 | 268.033 |
| **CM-7** | 0.979 | 0.967 | 0.076 | 0.067 | 0.085 | 227.553 |
| **CM-8** | 0.979 | 0.967 | 0.080 | 0.071 | 0.089 | 247.474 |
| **CM-9** | 0.975 | 0.960 | 0.084 | 0.075 | 0.093 | 270.736 |
| **CM-10** | 0.967 | 0.949 | 0.090 | 0.081 | 0.099 | 307.232 |
| **CM1-10** | Pooled SRMR: 0.057 | | | | | |
| **SIM-1** | 0.975 | 0.974 | 0.066 | 0.058 | 0.073 | 270.276 |
| **SIM-2** | 0.971 | 0.971 | 0.075 | 0.067 | 0.082 | 335.870 |
| **SIM-3** | 0.980 | 0.979 | 0.072 | 0.065 | 0.079 | 313.837 |
| **SIM-4** | 0.972 | 0.971 | 0.073 | 0.065 | 0.080 | 319.411 |
| **SIM-5** | 0.977 | 0.976 | 0.070 | 0.063 | 0.078 | 301.303 |
| **SIM-6** | 0.973 | 0.972 | 0.069 | 0.061 | 0.076 | 290.286 |
| **SIM-7** | 0.976 | 0.975 | 0.066 | 0.058 | 0.073 | 268.221 |
| **SIM-8** | 0.973 | 0.972 | 0.073 | 0.065 | 0.080 | 319.566 |
| **SIM-9** | 0.973 | 0.972 | 0.071 | 0.063 | 0.078 | 303.678 |
| **SIM-10** | 0.966 | 0.965 | 0.074 | 0.067 | 0.082 | 331.894 |
| **SIM1-10** | **Pooled SRMR: 0.057** | | | | | |
| **FIM-1** | 0.975 | 0.978 | 0.061 | 0.054 | 0.068 | 275.396 |
| **FIM-2** | 0.968 | 0.972 | 0.073 | 0.066 | 0.080 | 374.584 |
| **FIM-3** | 0.970 | 0.974 | 0.081 | 0.074 | 0.088 | 448.215 |
| **FIM-4** | 0.969 | 0.972 | 0.071 | 0.064 | 0.078 | 358.451 |
| **FIM-5** | 0.974 | 0.977 | 0.068 | 0.061 | 0.075 | 333.487 |
| **FIM-6** | 0.974 | 0.977 | 0.063 | 0.056 | 0.070 | 278.475 |
| **FIM-7** | 0.978 | 0.981 | 0.058 | 0.051 | 0.065 | 255.501 |
| **FIM-8** | 0.975 | 0.978 | 0.065 | 0.058 | 0.072 | 304.653 |

| | | | | | | |
|---|---|---|---|---|---|---|
| **FIM-9** | 0.971 | 0.974 | 0.067 | 0.061 | 0.075 | 326.333 |
| **FIM-10** | 0.968 | 0.971 | 0.067 | 0.060 | 0.074 | 323.691 |
| **FIM1-10** | Pooled SRMR: 0.060 | | | | | |
| *Family support (Epstein et al., 1983), 5 items, 0 unique item covariances* | | | | | | |
| **CM-1** | 0.990 | 0.985 | 0.057 | 0.047 | 0.066 | 138.928 |
| **CM-2** | 0.992 | 0.987 | 0.052 | 0.043 | 0.062 | 122.789 |
| **CM-3** | 0.994 | 0.99 | 0.046 | 0.036 | 0.056 | 100.896 |
| **CM-4** | 0.993 | 0.99 | 0.046 | 0.037 | 0.056 | 102.619 |
| **CM-5** | 0.991 | 0.987 | 0.054 | 0.044 | 0.063 | 128.006 |
| **CM-6** | 0.992 | 0.988 | 0.048 | 0.039 | 0.058 | 108.791 |
| **CM-7** | 0.990 | 0.985 | 0.056 | 0.047 | 0.066 | 138.048 |
| **CM-8** | 0.989 | 0.983 | 0.059 | 0.049 | 0.068 | 146.971 |
| **CM-9** | 0.991 | 0.986 | 0.052 | 0.043 | 0.062 | 123.619 |
| **CM-10** | 0.992 | 0.988 | 0.051 | 0.042 | 0.061 | 119.283 |
| **CM1-10** | Pooled SRMR: 0.029 | | | | | |
| **SIM-1** | 0.985 | 0.983 | 0.06 | 0.052 | 0.068 | 212.679 |
| **SIM-2** | 0.99 | 0.989 | 0.049 | 0.041 | 0.057 | 153.589 |
| **SIM-3** | 0.989 | 0.988 | 0.051 | 0.043 | 0.059 | 162.287 |
| **SIM-4** | 0.989 | 0.988 | 0.051 | 0.043 | 0.059 | 161.544 |
| **SIM-5** | 0.989 | 0.987 | 0.052 | 0.044 | 0.061 | 169.785 |
| **SIM-6** | 0.99 | 0.989 | 0.047 | 0.039 | 0.055 | 144.249 |
| **SIM-7** | 0.987 | 0.985 | 0.055 | 0.047 | 0.063 | 185.314 |
| **SIM-8** | 0.987 | 0.986 | 0.054 | 0.046 | 0.063 | 180.133 |
| **SIM-9** | 0.988 | 0.987 | 0.051 | 0.043 | 0.060 | 165.77 |
| **SIM-10** | 0.991 | 0.990 | 0.048 | 0.040 | 0.056 | 148.522 |
| **SIM1-10** | Pooled SRMR: 0.029 | | | | | |
| **FIM-1** | 0.981 | 0.981 | 0.063 | 0.056 | 0.071 | 269.238 |
| **FIM-2** | 0.984 | 0.985 | 0.056 | 0.049 | 0.063 | 221.745 |
| **FIM-3** | 0.981 | 0.981 | 0.063 | 0.056 | 0.071 | 271.247 |
| **FIM-4** | 0.984 | 0.984 | 0.057 | 0.05 | 0.064 | 227.918 |
| **FIM-5** | 0.985 | 0.986 | 0.055 | 0.048 | 0.063 | 218.267 |
| **FIM-6** | 0.989 | 0.989 | 0.046 | 0.039 | 0.054 | 166.872 |
| **FIM-7** | 0.983 | 0.984 | 0.057 | 0.05 | 0.065 | 230.402 |
| **FIM-8** | 0.985 | 0.986 | 0.054 | 0.046 | 0.061 | 207.083 |
| **FIM-9** | 0.988 | 0.989 | 0.048 | 0.040 | 0.055 | 174.514 |
| **FIM-10** | 0.982 | 0.983 | 0.061 | 0.054 | 0.069 | 257.422 |
| **FIM1-10** | Pooled SRMR: 0.041 | | | | | |
| *Family cohesion (Epstein et al., 1983), 7 items, 1 unique item covariance* | | | | | | |
| **CM-1** | 0.983 | 0.977 | 0.053 | 0.047 | 0.059 | 288.144 |
| **CM-2** | 0.97 | 0.959 | 0.066 | 0.06 | 0.072 | 414.681 |
| **CM-3** | 0.984 | 0.978 | 0.055 | 0.048 | 0.061 | 303.946 |
| **CM-4** | 0.97 | 0.959 | 0.071 | 0.065 | 0.077 | 470.339 |
| **CM-5** | 0.983 | 0.976 | 0.053 | 0.047 | 0.06 | 292.398 |
| **CM-6** | 0.98 | 0.973 | 0.056 | 0.05 | 0.062 | 314.174 |
| **CM-7** | 0.988 | 0.983 | 0.047 | 0.041 | 0.054 | 244.447 |
| **CM-8** | 0.98 | 0.973 | 0.056 | 0.05 | 0.063 | 318.451 |
| **CM-9** | 0.981 | 0.974 | 0.055 | 0.049 | 0.061 | 308.343 |
| **CM-10** | 0.985 | 0.979 | 0.052 | 0.046 | 0.058 | 280.724 |
| **CM1-10** | Pooled SRMR: 0.043 | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| **SIM-1** | 0.978 | 0.976 | 0.054 | 0.049 | 0.06 | 368.208 |
| **SIM-2** | 0.964 | 0.96 | 0.066 | 0.06 | 0.071 | 502.767 |
| **SIM-3** | 0.978 | 0.975 | 0.057 | 0.052 | 0.063 | 399.398 |
| **SIM-4** | 0.964 | 0.96 | 0.071 | 0.065 | 0.076 | 571.349 |
| **SIM-5** | 0.979 | 0.976 | 0.053 | 0.048 | 0.059 | 358.802 |
| **SIM-6** | 0.975 | 0.973 | 0.056 | 0.051 | 0.062 | 391.492 |
| **SIM-7** | 0.98 | 0.978 | 0.055 | 0.049 | 0.06 | 374.379 |
| **SIM-8** | 0.975 | 0.972 | 0.057 | 0.051 | 0.063 | 398.621 |
| **SIM-9** | 0.977 | 0.974 | 0.055 | 0.05 | 0.061 | 381.047 |
| **SIM-10** | 0.981 | 0.979 | 0.052 | 0.047 | 0.058 | 348.549 |
| **SIM1-10** | Pooled SRMR: 0.044 | | | | | |
| **FIM-1** | 0.969 | 0.969 | 0.061 | 0.056 | 0.067 | 499.191 |
| **FIM-2** | 0.964 | 0.964 | 0.062 | 0.057 | 0.067 | 506.42 |
| **FIM-3** | 0.962 | 0.962 | 0.071 | 0.066 | 0.076 | 631.775 |
| **FIM-4** | 0.959 | 0.959 | 0.071 | 0.066 | 0.076 | 639.074 |
| **FIM-5** | 0.973 | 0.973 | 0.057 | 0.052 | 0.063 | 443.698 |
| **FIM-6** | 0.967 | 0.967 | 0.062 | 0.057 | 0.068 | 511.904 |
| **FIM-7** | 0.963 | 0.963 | 0.071 | 0.065 | 0.076 | 627.937 |
| **FIM-8** | 0.965 | 0.965 | 0.063 | 0.058 | 0.069 | 525.539 |
| **FIM-9** | 0.973 | 0.973 | 0.057 | 0.052 | 0.063 | 443.87 |
| **FIM-10** | 0.966 | 0.966 | 0.066 | 0.061 | 0.071 | 563.688 |
| **FIM1-10** | Pooled SRMR: 0.055 | | | | | |
| *Positive self-esteem (Rosenberg, 1965), 5 items, 0 unique item covariances* | | | | | | |
| **CM-1** | 0.994 | 0.99 | 0.069 | 0.06 | 0.079 | 194.745 |
| **CM-2** | 0.995 | 0.993 | 0.059 | 0.05 | 0.068 | 148.122 |
| **CM-3** | 0.996 | 0.994 | 0.055 | 0.046 | 0.064 | 132.42 |
| **CM-4** | 0.996 | 0.994 | 0.056 | 0.047 | 0.066 | 137.011 |
| **CM-5** | 0.997 | 0.995 | 0.052 | 0.042 | 0.061 | 121.014 |
| **CM-6** | 0.996 | 0.993 | 0.057 | 0.048 | 0.067 | 141.289 |
| **CM-7** | 0.997 | 0.995 | 0.05 | 0.04 | 0.059 | 113.795 |
| **CM-8** | 0.997 | 0.995 | 0.051 | 0.042 | 0.061 | 118.093 |
| **CM-9** | 0.996 | 0.994 | 0.055 | 0.046 | 0.065 | 134.281 |
| **CM-10** | 0.996 | 0.994 | 0.055 | 0.046 | 0.065 | 133.806 |
| **CM1-10** | Pooled SRMR: 0.022 | | | | | |
| **SIM-1** | 0.993 | 0.992 | 0.062 | 0.055 | 0.07 | 234.615 |
| **SIM-2** | 0.994 | 0.994 | 0.054 | 0.047 | 0.062 | 189.538 |
| **SIM-3** | 0.995 | 0.995 | 0.05 | 0.043 | 0.059 | 168.888 |
| **SIM-4** | 0.995 | 0.995 | 0.053 | 0.045 | 0.061 | 182.032 |
| **SIM-5** | 0.996 | 0.995 | 0.05 | 0.042 | 0.058 | 167.425 |
| **SIM-6** | 0.996 | 0.995 | 0.049 | 0.041 | 0.057 | 159.632 |
| **SIM-7** | 0.996 | 0.996 | 0.044 | 0.036 | 0.053 | 140.022 |
| **SIM-8** | 0.996 | 0.995 | 0.047 | 0.039 | 0.055 | 152.146 |
| **SIM-9** | 0.996 | 0.996 | 0.047 | 0.039 | 0.055 | 150.135 |
| **SIM-10** | 0.995 | 0.995 | 0.051 | 0.043 | 0.059 | 171.582 |
| **SIM1-10** | Pooled SRMR: 0.022 | | | | | |
| **FIM-1** | 0.993 | 0.994 | 0.054 | 0.047 | 0.062 | 219.431 |
| **FIM-2** | 0.994 | 0.994 | 0.053 | 0.045 | 0.06 | 210.452 |
| **FIM-3** | 0.994 | 0.995 | 0.053 | 0.046 | 0.06 | 212.1 |
| **FIM-4** | 0.993 | 0.994 | 0.058 | 0.051 | 0.065 | 243.325 |
| **FIM-5** | 0.993 | 0.993 | 0.06 | 0.053 | 0.068 | 260.99 |

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|---|---|---|---|---|---|---|
| **FIM-6** | 0.995 | 0.995 | 0.048 | 0.041 | 0.055 | 182.094 |
| **FIM-7** | 0.994 | 0.994 | 0.054 | 0.047 | 0.062 | 219.7 |
| **FIM-8** | 0.995 | 0.996 | 0.045 | 0.037 | 0.052 | 165.949 |
| **FIM-9** | 0.996 | 0.996 | 0.044 | 0.037 | 0.052 | 162.66 |
| **FIM-10** | 0.994 | 0.994 | 0.054 | 0.047 | 0.061 | 216.742 |
| **FIM1-10** | Pooled SRMR: 0.029 | | | | | |
| *Negative self-esteem (Rosenberg, 1965), 5 items, 0 unique item covariances* | | | | | | |
| **CM-1** | 0.999 | 0.999 | 0.025 | 0.013 | 0.036 | 50.578 |
| **CM-2** | 0.999 | 0.999 | 0.025 | 0.013 | 0.036 | 50.643 |
| **CM-3** | 0.999 | 0.999 | 0.025 | 0.012 | 0.036 | 50.203 |
| **CM-4** | 1 | 0.999 | 0.023 | 0.01 | 0.035 | 47.206 |
| **CM-5** | 1 | 0.999 | 0.02 | 0 | 0.032 | 42.357 |
| **CM-6** | 1 | 0.999 | 0.02 | 0.003 | 0.032 | 43.016 |
| **CM-7** | 1 | 1 | 0.015 | 0 | 0.028 | 36.877 |
| **CM-8** | 0.999 | 0.999 | 0.024 | 0.012 | 0.036 | 49.542 |
| **CM-9** | 0.999 | 0.999 | 0.027 | 0.015 | 0.038 | 54.188 |
| **CM-10** | 1 | 1 | 0.018 | 0 | 0.03 | 40.01 |
| **CM1-10** | Pooled SRMR: 0.023 | | | | | |
| **SIM-1** | 0.999 | 0.999 | 0.031 | 0.022 | 0.04 | 89.463 |
| **SIM-2** | 0.999 | 0.999 | 0.031 | 0.022 | 0.04 | 88.917 |
| **SIM-3** | 0.999 | 0.999 | 0.028 | 0.018 | 0.037 | 80.439 |
| **SIM-4** | 0.999 | 0.999 | 0.024 | 0.013 | 0.033 | 70.227 |
| **SIM-5** | 0.999 | 0.999 | 0.023 | 0.012 | 0.033 | 68.249 |
| **SIM-6** | 0.999 | 0.999 | 0.024 | 0.014 | 0.033 | 70.689 |
| **SIM-7** | 0.999 | 0.999 | 0.024 | 0.013 | 0.033 | 69.754 |
| **SIM-8** | 0.999 | 0.999 | 0.03 | 0.021 | 0.039 | 87.168 |
| **SIM-9** | 0.999 | 0.999 | 0.028 | 0.019 | 0.037 | 80.832 |
| **SIM-10** | 0.999 | 0.999 | 0.021 | 0.009 | 0.031 | 63.684 |
| **SIM1-10** | Pooled SRMR: 0.023 | | | | | |
| **FIM-1** | 0.989 | 0.989 | 0.084 | 0.078 | 0.092 | 463.987 |
| **FIM-2** | 0.988 | 0.989 | 0.088 | 0.081 | 0.095 | 503.638 |
| **FIM-3** | 0.985 | 0.986 | 0.091 | 0.084 | 0.098 | 527.52 |
| **FIM-4** | 0.986 | 0.987 | 0.094 | 0.087 | 0.101 | 559.567 |
| **FIM-5** | 0.986 | 0.987 | 0.094 | 0.088 | 0.102 | 567.829 |
| **FIM-6** | 0.986 | 0.987 | 0.09 | 0.084 | 0.098 | 525.236 |
| **FIM-7** | 0.989 | 0.989 | 0.089 | 0.082 | 0.096 | 506.841 |
| **FIM-8** | 0.988 | 0.989 | 0.086 | 0.079 | 0.093 | 474.214 |
| **FIM-9** | 0.984 | 0.985 | 0.104 | 0.097 | 0.111 | 678.34 |
| **FIM-10** | 0.986 | 0.987 | 0.094 | 0.087 | 0.101 | 557.688 |
| **FIM1-10** | Pooled SRMR: 0.063 | | | | | |
| *Brooding old (Burwell & Shirk, 2007; Treynor et al., 2003), 4 items, 0 unique item covariances* | | | | | | |
| **CM-1** | 0.977 | 0.956 | 0.067 | 0.054 | 0.08 | 93.868 |
| **CM-2** | 0.971 | 0.947 | 0.078 | 0.065 | 0.091 | 122.255 |
| **CM-3** | 0.977 | 0.957 | 0.068 | 0.055 | 0.081 | 96.623 |
| **CM-4** | 0.98 | 0.962 | 0.071 | 0.059 | 0.084 | 105.314 |
| **CM-5** | 0.977 | 0.956 | 0.076 | 0.063 | 0.089 | 117.05 |
| **CM-6** | 0.973 | 0.949 | 0.078 | 0.066 | 0.091 | 123.139 |
| **CM-7** | 0.977 | 0.957 | 0.069 | 0.056 | 0.082 | 98.864 |
| **CM-8** | 0.973 | 0.949 | 0.071 | 0.058 | 0.084 | 104.333 |

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|---|---|---|---|---|---|---|
| **CM-9** | 0.969 | 0.942 | 0.077 | 0.065 | 0.09 | 121.504 |
| **CM-10** | 0.971 | 0.946 | 0.078 | 0.065 | 0.091 | 122.079 |
| **CM1-10** | Pooled SRMR: 0.054 | | | | | |
| **SIM-1** | 0.936 | 0.929 | 0.085 | 0.075 | 0.095 | 238.995 |
| **SIM-2** | 0.927 | 0.918 | 0.096 | 0.086 | 0.106 | 298.739 |
| **SIM-3** | 0.936 | 0.928 | 0.088 | 0.078 | 0.098 | 252.866 |
| **SIM-4** | 0.937 | 0.93 | 0.097 | 0.087 | 0.107 | 303.33 |
| **SIM-5** | 0.935 | 0.927 | 0.098 | 0.088 | 0.107 | 307.155 |
| **SIM-6** | 0.932 | 0.924 | 0.096 | 0.086 | 0.106 | 296.329 |
| **SIM-7** | 0.936 | 0.928 | 0.089 | 0.08 | 0.099 | 261.496 |
| **SIM-8** | 0.93 | 0.922 | 0.088 | 0.078 | 0.098 | 254.419 |
| **SIM-9** | 0.938 | 0.93 | 0.085 | 0.075 | 0.095 | 237.518 |
| **SIM-10** | 0.928 | 0.919 | 0.095 | 0.085 | 0.105 | 291.417 |
| **SIM1-10** | Pooled SRMR: 0.062 | | | | | |
| **FIM-1** | 0.942 | 0.948 | 0.073 | 0.064 | 0.082 | 225.822 |
| **FIM-2** | 0.934 | 0.94 | 0.082 | 0.074 | 0.091 | 279.996 |
| **FIM-3** | 0.946 | 0.952 | 0.072 | 0.063 | 0.081 | 221.084 |
| **FIM-4** | 0.941 | 0.947 | 0.084 | 0.076 | 0.093 | 292.813 |
| **FIM-5** | 0.939 | 0.945 | 0.085 | 0.076 | 0.094 | 296.591 |
| **FIM-6** | 0.937 | 0.943 | 0.082 | 0.074 | 0.091 | 280.363 |
| **FIM-7** | 0.941 | 0.947 | 0.077 | 0.068 | 0.086 | 247.179 |
| **FIM-8** | 0.939 | 0.945 | 0.074 | 0.065 | 0.083 | 231.274 |
| **FIM-9** | 0.942 | 0.948 | 0.073 | 0.064 | 0.082 | 227.791 |
| **FIM-10** | 0.937 | 0.943 | 0.08 | 0.071 | 0.089 | 265.313 |
| **FIM1-10** | Pooled SRMR: 0.064 | | | | | |
| *Brooding new (Burwell & Shirk, 2007; Treynor et al., 2003), 5 items, 0 unique item covariances* | | | | | | |
| **CM-1** | 0.992 | 0.988 | 0.048 | 0.038 | 0.057 | 106.843 |
| **CM-2** | 0.992 | 0.987 | 0.048 | 0.038 | 0.058 | 107.93 |
| **CM-3** | 0.991 | 0.986 | 0.05 | 0.04 | 0.059 | 113.783 |
| **CM-4** | 0.992 | 0.988 | 0.048 | 0.039 | 0.058 | 108.199 |
| **CM-5** | 0.991 | 0.986 | 0.05 | 0.041 | 0.06 | 115.891 |
| **CM-6** | 0.992 | 0.988 | 0.047 | 0.037 | 0.057 | 104.157 |
| **CM-7** | 0.993 | 0.99 | 0.043 | 0.033 | 0.053 | 92.914 |
| **CM-8** | 0.993 | 0.988 | 0.045 | 0.035 | 0.055 | 98.435 |
| **CM-9** | 0.992 | 0.988 | 0.046 | 0.036 | 0.056 | 101.432 |
| **CM-10** | 0.993 | 0.989 | 0.044 | 0.034 | 0.054 | 94.903 |
| **CM1-10** | Pooled SRMR: 0.032 | | | | | |
| **SIM-1** | 0.99 | 0.989 | 0.045 | 0.037 | 0.053 | 142.215 |
| **SIM-2** | 0.99 | 0.99 | 0.043 | 0.035 | 0.051 | 133.586 |
| **SIM-3** | 0.988 | 0.987 | 0.047 | 0.039 | 0.056 | 153.827 |
| **SIM-4** | 0.99 | 0.99 | 0.044 | 0.036 | 0.052 | 138.354 |
| **SIM-5** | 0.988 | 0.987 | 0.048 | 0.04 | 0.056 | 156.958 |
| **SIM-6** | 0.991 | 0.99 | 0.043 | 0.035 | 0.051 | 133.694 |
| **SIM-7** | 0.99 | 0.99 | 0.043 | 0.035 | 0.052 | 135.477 |
| **SIM-8** | 0.991 | 0.99 | 0.042 | 0.034 | 0.05 | 129.786 |
| **SIM-9** | 0.99 | 0.989 | 0.043 | 0.035 | 0.052 | 135.846 |
| **SIM-10** | 0.99 | 0.99 | 0.043 | 0.035 | 0.051 | 132.763 |
| **SIM1-10** | Pooled SRMR: 0.031 | | | | | |
| **FIM-1** | 0.986 | 0.987 | 0.049 | 0.041 | 0.056 | 186.199 |
| **FIM-2** | 0.981 | 0.983 | 0.055 | 0.048 | 0.063 | 227.768 |

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|---|---|---|---|---|---|---|
| **FIM-3** | 0.981 | 0.983 | 0.055 | 0.048 | 0.062 | 224.08 |
| **FIM-4** | 0.984 | 0.985 | 0.052 | 0.045 | 0.059 | 205.898 |
| **FIM-5** | 0.981 | 0.983 | 0.056 | 0.049 | 0.064 | 233.487 |
| **FIM-6** | 0.983 | 0.984 | 0.054 | 0.047 | 0.061 | 217.252 |
| **FIM-7** | 0.983 | 0.985 | 0.053 | 0.046 | 0.06 | 211.592 |
| **FIM-8** | 0.982 | 0.983 | 0.054 | 0.047 | 0.061 | 218.171 |
| **FIM-9** | 0.983 | 0.984 | 0.053 | 0.046 | 0.06 | 210.824 |
| **FIM-10** | 0.983 | 0.984 | 0.053 | 0.045 | 0.06 | 209.935 |
| **FIM1-10** | Pooled SRMR: 0.042 | | | | | |
| *Reflection (Burwell & Shirk, 2007; Treynor et al., 2003), 5 items, 1 unique item covariance* | | | | | | |
| **CM-1** | 0.992 | 0.986 | 0.046 | 0.037 | 0.057 | 95.986 |
| **CM-2** | 0.991 | 0.985 | 0.048 | 0.038 | 0.058 | 101.052 |
| **CM-3** | 0.99 | 0.983 | 0.05 | 0.04 | 0.06 | 105.905 |
| **CM-4** | 0.992 | 0.987 | 0.046 | 0.036 | 0.057 | 95.777 |
| **CM-5** | 0.992 | 0.986 | 0.047 | 0.037 | 0.057 | 98.052 |
| **CM-6** | 0.992 | 0.987 | 0.045 | 0.036 | 0.056 | 93.022 |
| **CM-7** | 0.994 | 0.989 | 0.041 | 0.031 | 0.052 | 81.375 |
| **CM-8** | 0.992 | 0.986 | 0.046 | 0.036 | 0.056 | 94.02 |
| **CM-9** | 0.992 | 0.986 | 0.048 | 0.038 | 0.058 | 99.497 |
| **CM-10** | 0.991 | 0.986 | 0.047 | 0.038 | 0.058 | 98.857 |
| **CM1-10** | Pooled SRMR: 0.045 | | | | | |
| **SIM-1** | 0.986 | 0.984 | 0.05 | 0.042 | 0.058 | 156.491 |
| **SIM-2** | 0.985 | 0.984 | 0.05 | 0.042 | 0.059 | 159.87 |
| **SIM-3** | 0.985 | 0.983 | 0.051 | 0.043 | 0.059 | 161.732 |
| **SIM-4** | 0.985 | 0.983 | 0.052 | 0.044 | 0.061 | 170.309 |
| **SIM-5** | 0.986 | 0.984 | 0.05 | 0.042 | 0.059 | 160.03 |
| **SIM-6** | 0.986 | 0.984 | 0.05 | 0.042 | 0.058 | 157.737 |
| **SIM-7** | 0.988 | 0.986 | 0.046 | 0.038 | 0.055 | 141.928 |
| **SIM-8** | 0.986 | 0.984 | 0.049 | 0.041 | 0.057 | 153.663 |
| **SIM-9** | 0.986 | 0.984 | 0.051 | 0.043 | 0.059 | 163.984 |
| **SIM-10** | 0.986 | 0.984 | 0.05 | 0.042 | 0.058 | 158.201 |
| **SIM1-10** | Pooled SRMR: 0.045 | | | | | |
| **FIM-1** | 0.966 | 0.968 | 0.071 | 0.064 | 0.079 | 330.033 |
| **FIM-2** | 0.968 | 0.969 | 0.069 | 0.062 | 0.076 | 313.059 |
| **FIM-3** | 0.963 | 0.965 | 0.073 | 0.065 | 0.08 | 340.539 |
| **FIM-4** | 0.967 | 0.968 | 0.072 | 0.065 | 0.079 | 336.733 |
| **FIM-5** | 0.968 | 0.969 | 0.07 | 0.063 | 0.077 | 319.366 |
| **FIM-6** | 0.97 | 0.971 | 0.067 | 0.06 | 0.074 | 298.213 |
| **FIM-7** | 0.969 | 0.97 | 0.069 | 0.062 | 0.076 | 311.317 |
| **FIM-8** | 0.967 | 0.968 | 0.069 | 0.062 | 0.077 | 315.391 |
| **FIM-9** | 0.967 | 0.969 | 0.071 | 0.064 | 0.078 | 327.705 |
| **FIM-10** | 0.965 | 0.966 | 0.072 | 0.065 | 0.079 | 336.78 |
| **FIM1-10** | Pooled SRMR: 0.063 | | | | | |
| *Distress tolerance (Bould et al., 2013), 5 items, 1 unique item covariance* | | | | | | |
| **CM-1** | 0.968 | 0.947 | 0.101 | 0.092 | 0.111 | 356.552 |
| **CM-2** | 0.969 | 0.948 | 0.105 | 0.096 | 0.114 | 379.225 |
| **CM-3** | 0.967 | 0.946 | 0.105 | 0.095 | 0.114 | 377.534 |
| **CM-4** | 0.968 | 0.947 | 0.103 | 0.094 | 0.113 | 369.052 |
| **CM-5** | 0.97 | 0.95 | 0.102 | 0.092 | 0.111 | 358.546 |

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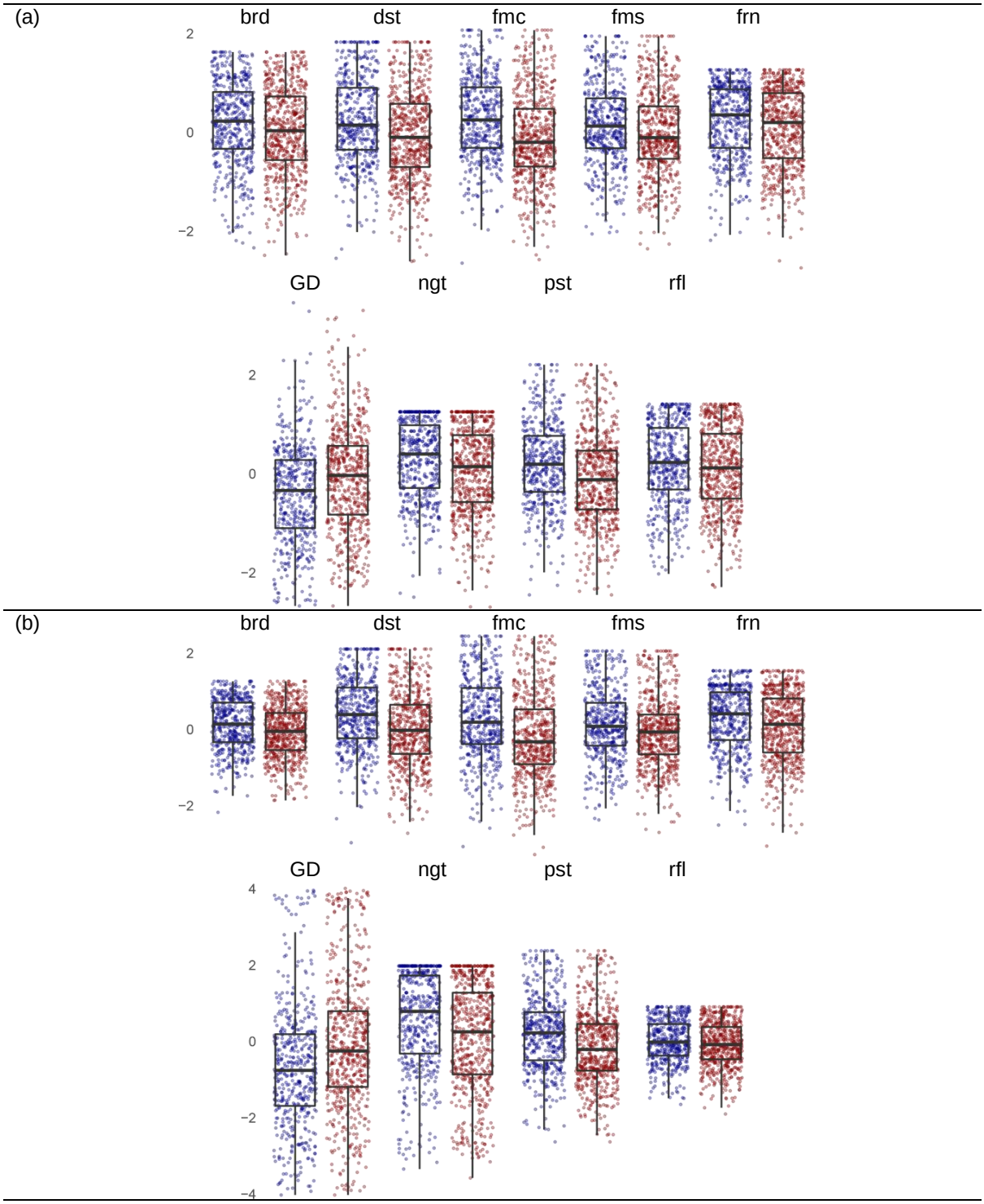
|---|---|---|---|---|---|---|
| **CM-6** | 0.971 | 0.951 | 0.101 | 0.092 | 0.111 | 354.518 |
| **CM-7** | 0.97 | 0.95 | 0.1 | 0.091 | 0.109 | 346.656 |
| **CM-8** | 0.972 | 0.954 | 0.097 | 0.088 | 0.107 | 329.16 |
| **CM-9** | 0.972 | 0.953 | 0.102 | 0.093 | 0.111 | 360.153 |
| **CM-10** | 0.97 | 0.95 | 0.101 | 0.092 | 0.11 | 353.137 |
| **CM1-10** | Pooled SRMR: 0.059 | | | | | |
| **SIM-1** | 0.968 | 0.968 | 0.079 | 0.072 | 0.086 | 376.783 |
| **SIM-2** | 0.97 | 0.97 | 0.079 | 0.072 | 0.086 | 378.301 |
| **SIM-3** | 0.966 | 0.966 | 0.083 | 0.076 | 0.09 | 411.1 |
| **SIM-4** | 0.969 | 0.969 | 0.079 | 0.072 | 0.087 | 379.576 |
| **SIM-5** | 0.97 | 0.97 | 0.079 | 0.072 | 0.086 | 377.378 |
| **SIM-6** | 0.97 | 0.97 | 0.079 | 0.072 | 0.086 | 377.037 |
| **SIM-7** | 0.97 | 0.97 | 0.077 | 0.07 | 0.085 | 364.221 |
| **SIM-8** | 0.973 | 0.973 | 0.074 | 0.067 | 0.082 | 337.75 |
| **SIM-9** | 0.971 | 0.971 | 0.079 | 0.072 | 0.087 | 382.255 |
| **SIM-10** | 0.969 | 0.969 | 0.08 | 0.072 | 0.087 | 383.474 |
| **SIM1-10** | Pooled SRMR: 0.060 | | | | | |
| **FIM-1** | 0.970 | 0.974 | 0.071 | 0.064 | 0.078 | 361.965 |
| **FIM-2** | 0.975 | 0.978 | 0.068 | 0.061 | 0.075 | 338.295 |
| **FIM-3** | 0.969 | 0.973 | 0.073 | 0.066 | 0.08 | 381.179 |
| **FIM-4** | 0.973 | 0.977 | 0.068 | 0.061 | 0.075 | 338.4 |
| **FIM-5** | 0.973 | 0.977 | 0.07 | 0.063 | 0.077 | 351.802 |
| **FIM-6** | 0.974 | 0.977 | 0.069 | 0.062 | 0.076 | 344.05 |
| **FIM-7** | 0.971 | 0.975 | 0.07 | 0.064 | 0.077 | 358.149 |
| **FIM-8** | 0.978 | 0.981 | 0.063 | 0.056 | 0.07 | 295.438 |
| **FIM-9** | 0.974 | 0.978 | 0.07 | 0.063 | 0.077 | 355.542 |
| **FIM-10** | 0.975 | 0.978 | 0.067 | 0.06 | 0.074 | 328.527 |
| **FIM1-10** | Pooled SRMR: 0.064 | | | | | |
| *Aggression (Goodyer et al., 2011), 4 items, 0 unique item covariances* | | | | | | |
| **CM-1** | 0.999 | 0.997 | 0.036 | 0.022 | 0.051 | 38.412 |
| **CM-2** | 0.998 | 0.995 | 0.052 | 0.039 | 0.065 | 62.624 |
| **CM-3** | 0.999 | 0.998 | 0.035 | 0.021 | 0.05 | 36.937 |
| **CM-4** | 0.997 | 0.994 | 0.057 | 0.044 | 0.07 | 72.567 |
| **CM-5** | 0.996 | 0.992 | 0.051 | 0.038 | 0.064 | 61.034 |
| **CM-6** | 0.999 | 0.998 | 0.032 | 0.017 | 0.047 | 33.314 |
| **CM-7** | 0.998 | 0.997 | 0.055 | 0.042 | 0.069 | 69.342 |
| **CM-8** | 0.998 | 0.996 | 0.046 | 0.033 | 0.06 | 53.348 |
| **CM-9** | 0.998 | 0.996 | 0.045 | 0.031 | 0.058 | 50.375 |
| **CM-10** | 0.999 | 0.997 | 0.04 | 0.026 | 0.054 | 43.08 |
| **CM1-10** | Pooled SRMR: 0.052 | | | | | |
| **SIM-1** | 0.993 | 0.992 | 0.063 | 0.053 | 0.073 | 136.864 |
| **SIM-2** | 0.993 | 0.991 | 0.071 | 0.061 | 0.081 | 167.668 |
| **SIM-3** | 0.994 | 0.993 | 0.058 | 0.048 | 0.068 | 119.667 |
| **SIM-4** | 0.992 | 0.991 | 0.071 | 0.061 | 0.081 | 167.166 |
| **SIM-5** | 0.989 | 0.988 | 0.065 | 0.055 | 0.075 | 144.604 |
| **SIM-6** | 0.994 | 0.993 | 0.059 | 0.049 | 0.069 | 122.171 |
| **SIM-7** | 0.995 | 0.994 | 0.074 | 0.064 | 0.084 | 179.659 |
| **SIM-8** | 0.994 | 0.993 | 0.063 | 0.053 | 0.073 | 136.06 |
| **SIM-9** | 0.992 | 0.991 | 0.068 | 0.058 | 0.079 | 157.416 |
| **SIM-10** | 0.994 | 0.993 | 0.062 | 0.052 | 0.073 | 134.713 |

| | | | | | | |
|---|---|---|---|---|---|---|
| **SIM1-10** | Pooled SRMR: 0.076 | | | | | |
| **FIM-1** | 0.993 | 0.993 | 0.059 | 0.05 | 0.069 | 155.276 |
| **FIM-2** | 0.993 | 0.994 | 0.061 | 0.052 | 0.07 | 161.385 |
| **FIM-3** | 0.993 | 0.993 | 0.059 | 0.05 | 0.068 | 154.017 |
| **FIM-4** | 0.993 | 0.993 | 0.061 | 0.052 | 0.07 | 162.482 |
| **FIM-5** | 0.99 | 0.99 | 0.058 | 0.049 | 0.067 | 148.267 |
| **FIM-6** | 0.992 | 0.992 | 0.06 | 0.051 | 0.07 | 159.709 |
| **FIM-7** | 0.994 | 0.994 | 0.073 | 0.064 | 0.083 | 221.604 |
| **FIM-8** | 0.994 | 0.994 | 0.058 | 0.049 | 0.067 | 148.019 |
| **FIM-9** | 0.992 | 0.993 | 0.062 | 0.053 | 0.071 | 166.672 |
| **FIM-10** | 0.993 | 0.993 | 0.063 | 0.054 | 0.073 | 172.324 |
| **FIM1-10** | Pooled SRMR: 0.119 | | | | | |
| *General distress (Messer et al., 1995; Reynolds & Richmond, 1978), 41 items, 2 unique item covariances* | | | | | | |
| **CM-1*** | 0.989 | 0.988 | 0.026 | 0.025 | 0.027 | 5752.268 |
| **CM-2*** | 0.989 | 0.988 | 0.026 | 0.025 | 0.027 | 5784.608 |
| **CM-3*** | 0.987 | 0.987 | 0.026 | 0.025 | 0.027 | 5707.489 |
| **CM-4*** | 0.989 | 0.989 | 0.026 | 0.025 | 0.027 | 5728.317 |
| **CM-5*** | 0.989 | 0.988 | 0.026 | 0.025 | 0.027 | 5795.843 |
| **CM-6*** | 0.987 | 0.987 | 0.027 | 0.026 | 0.028 | 5900.122 |
| **CM-7*** | 0.990 | 0.989 | 0.026 | 0.025 | 0.027 | 5787.842 |
| **CM-8*** | 0.989 | 0.989 | 0.026 | 0.025 | 0.027 | 5723.574 |
| **CM-9*** | 0.988 | 0.987 | 0.027 | 0.026 | 0.028 | 5892.805 |
| **CM-10*** | 0.987 | 0.987 | 0.027 | 0.026 | 0.028 | 5981.058 |
| **CM1-10** | Pooled SRMR: 0.044 | | | | | |
| **SIM-1** | 0.987 | 0.987 | 0.027 | 0.026 | 0.028 | 6211.303 |
| **SIM-2** | 0.987 | 0.987 | 0.027 | 0.026 | 0.028 | 6249.019 |
| **SIM-3** | 0.986 | 0.986 | 0.027 | 0.026 | 0.028 | 6137.497 |
| **SIM-4** | 0.988 | 0.988 | 0.027 | 0.026 | 0.028 | 6173.344 |
| **SIM-5** | 0.987 | 0.987 | 0.027 | 0.026 | 0.028 | 6217.918 |
| **SIM-6** | 0.986 | 0.986 | 0.028 | 0.027 | 0.029 | 6323.823 |
| **SIM-7** | 0.988 | 0.988 | 0.027 | 0.026 | 0.028 | 6273.396 |
| **SIM-8** | 0.988 | 0.988 | 0.027 | 0.026 | 0.028 | 6148.299 |
| **SIM-9** | 0.986 | 0.986 | 0.028 | 0.027 | 0.029 | 6341.883 |
| **SIM-10** | 0.986 | 0.986 | 0.028 | 0.027 | 0.029 | 6432.838 |
| **SIM1-10** | Pooled SRMR: 0.044 | | | | | |
| **FIM-1** | 0.953 | 0.953 | 0.052 | 0.051 | 0.053 | 14055.219 |
| **FIM-2** | 0.953 | 0.954 | 0.052 | 0.051 | 0.053 | 14248.743 |
| **FIM-3** | 0.949 | 0.949 | 0.051 | 0.050 | 0.052 | 13603.930 |
| **FIM-4** | 0.953 | 0.953 | 0.053 | 0.052 | 0.054 | 14707.235 |
| **FIM-5** | 0.952 | 0.953 | 0.053 | 0.052 | 0.053 | 14350.666 |
| **FIM-6** | 0.952 | 0.953 | 0.050 | 0.050 | 0.051 | 13508.845 |
| **FIM-7** | 0.953 | 0.953 | 0.055 | 0.054 | 0.056 | 15307.465 |
| **FIM-8** | 0.955 | 0.955 | 0.052 | 0.051 | 0.053 | 14073.361 |
| **FIM-9** | 0.951 | 0.952 | 0.052 | 0.051 | 0.053 | 14046.556 |
| **FIM-10** | 0.951 | 0.951 | 0.052 | 0.051 | 0.053 | 14007.992 |
| **FIM1-10** | Pooled SRMR: 0.107 | | | | | |

*Note.* WLSMV = weighted least squares estimator with mean- and variance corrected test statistics and robust standard errors. CFI = Comparative fit index, TLI = Tucker-Lewis index, RMSEA = Root mean square error of approximation, CI = Confidence interval, CM = configural model, SIM = strong invariance model, FIM = full invariance model. *For the configural model of the general distress factor, we had to enforce the loadings to be positive to ensure that they would not switch negative. We had to do this, as for some of the imputation data sets,

the loadings switched negative and when pooling over the coefficients the positive and negative loadings would have averaged each other out.



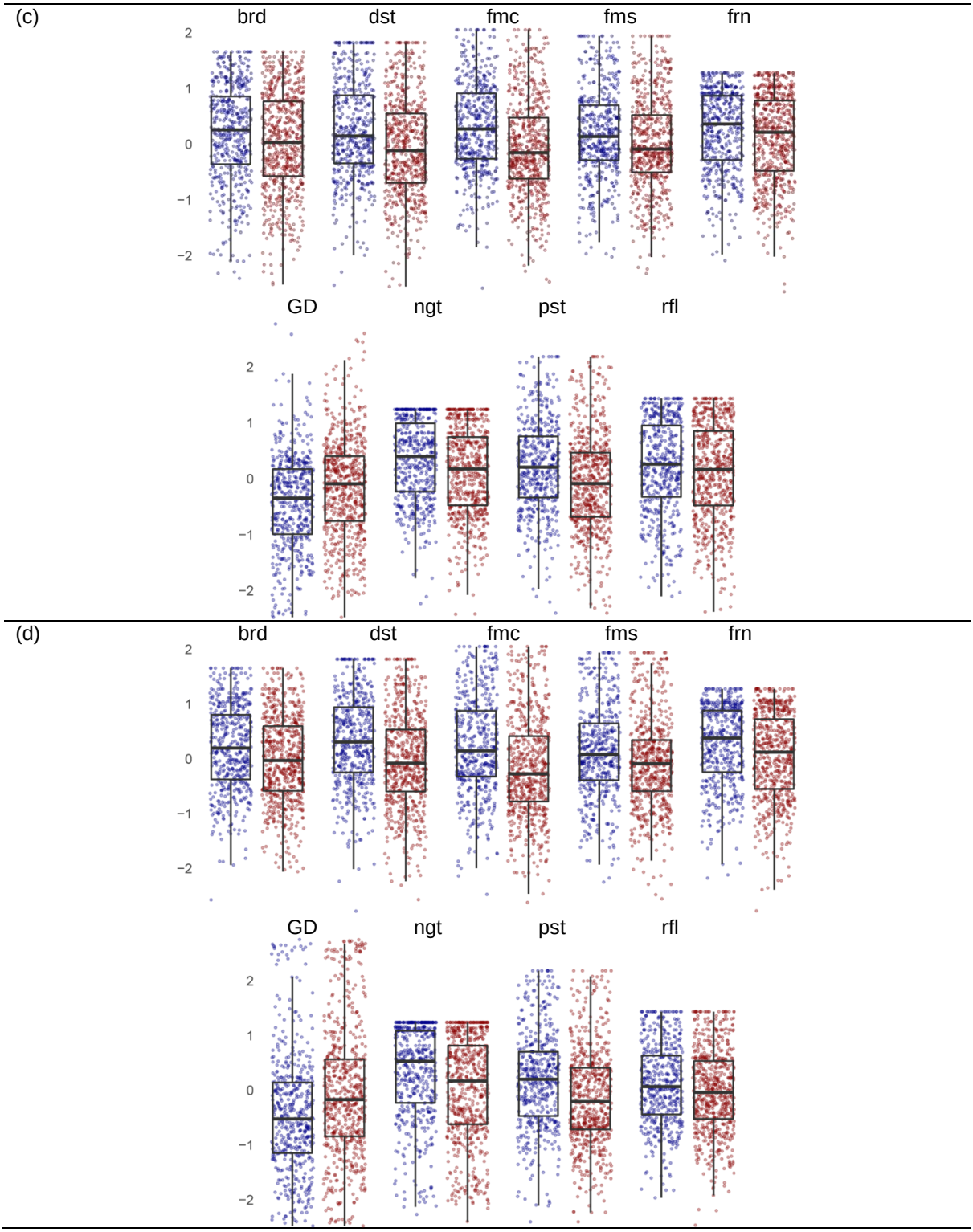



*Figure C.5.* Box-and-whisker plots with individual data points for the RFs (except expressive suppression and aggression) and the general distress variable, separately for CA+ (n = 631) and CA- (n = 499) groups. Panel (a) depicts the distributions for the strongly invariant scores for age 14 and panel (b) for age 17. Panel (c) depicts the distributions for the fully invariant scores for age 14 and panel (d) for age 17. CA- group = blue data points, CA+ group = red data points. Center line = median (50% quantile); lower box limit =25% quantile; upper box limit = 75% quantile; lower whisker = smallest observation greater than or equal to the lower box limit - 1.5 x Inter Quartile Range (IQR); upper whisker = largest observation less than or equal to upper box limit + 1.5 x IQR; outliers = data points beyond the end of the whiskers. **Legend:** Brd = brooding, dst = distress tolerance, fmc = family cohesion, fms = family support, frn = friend support, ngt = negative self-esteem, GD = general distress, pst = positive self-esteem, rfl = reflective rumination.

## APPENDIX C.6: MEAN CHANGE ANALYSES WITH (A) FULLY INVARIANT FACTOR SCORES AND (B) SUM SCORES

We compared the RF and general distress mean levels between age 14 and age 17, separately in the CA+ and CA- groups, based on strongly invariant factor scores, fully invariant factor scores, and sum scores. Results for the strongly invariant factor scores can be found in Chapter 4 (see also Figure 1 in Chapter 4). We decided that the strongly invariant model is the most adequate model for latent mean comparisons, as it is the least constrained one of the sufficiently constrained models.

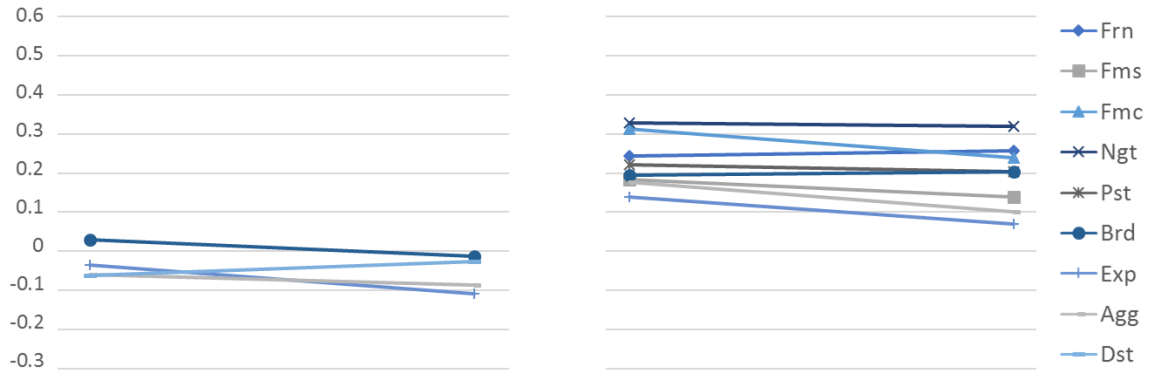
**Fully invariant factor scores.** For the fully invariant factor scores, the results looked somewhat different. This time, change was particularly notable in the CA+ group. In the CA- group only reflection and distress tolerance changed. While reflection deceased over time, distress tolerance increased over time, as for the strongly invariant scores. In the CA+ group instead of two this time six RFs changed between age 14 and 17. All inter-personal RFs (i.e. friendship support, family support and family cohesion) and three intra-personal RFs (negative self-esteem, positive self-esteem, and reflection) decreased between age 14 and 17 (see Figure C.6a). Of those six decreasing RFs in the CA+ group, only two reached a p-values below 0.025 (namely family cohesion and reflection). Importantly, none of the RFs changed significantly different in the CA+ and the CA- group (as tested with interaction effects), which indicates that the detected changes in the CA+ group, which were non-significant in the CA- group, were so minor that they did not differ significantly between the two groups. Of note, the binarized aggression and expressive suppression RFs are the same variables as reported in Chapter 4.

**Sum scores.** For the sum scores, the results looked again somewhat different from the strongly invariant factor scores. However, the change patterns were the same as for the fully invariant factor scores (see Figure C.6b). Importantly, once more none of the RFs changed significantly different in the CA+ and the CA- group (as tested with interaction effects). Hence, the same conclusion seems to hold as for fully invariant factor scores, namely that the detected changes in the CA+ group, which were non-significant in the CA- group, were so minor that they did not differ significantly between the two groups. Hence, overall we conclude that when investigating fully invariant factor scores and sum scores there seemed to be more variability in the CA+ group between age 14 and 17, yet, those changes were so minor that they did not differ between the two groups.

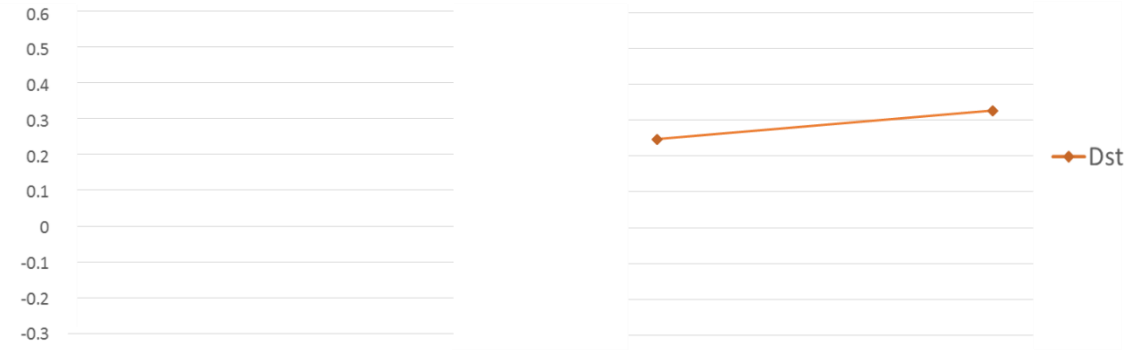
## A. Fully invariant factor scores

| CA+ | CA- |
|---|---|

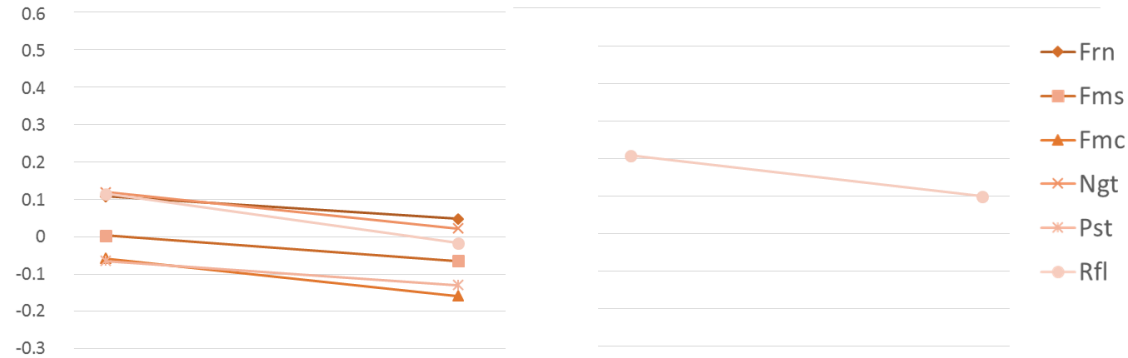
*No RF change*




*Increasing RFs*




*Decreasing RFs*



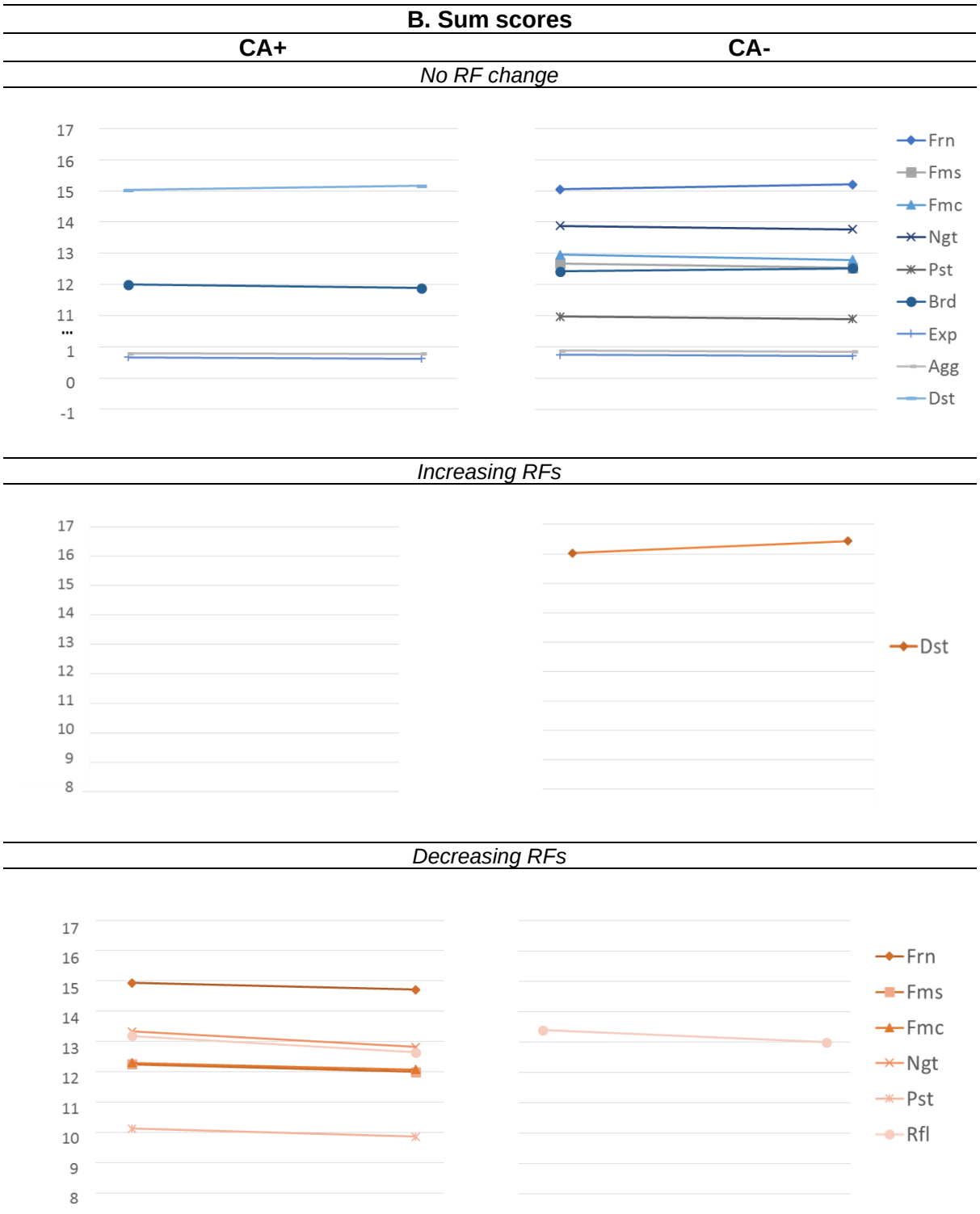



*Figure C.6.* RF and general distress mean level comparisons: age 14 versus age 17. CA = childhood adversity. Panel A scores are derived from fully invariant confirmatory factor analyses; panel B scores are sum scores. All RFs are scored in such a way that high values are protective (e.g. high levels of high friendship support or high levels of low negative self-esteem) and low values are harmful (e.g. low levels of high friendship support or low levels of low negative self-esteem). **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression.

## APPENDIX C.7: RESILIENCE FACTOR NETWORK RESULTS WITHOUT THE GENERAL DISTRESS VARIABLE AS WELL AS RESILIENCE FACTOR NETWORK RESULTS WITH THE GENERAL DISTRESS VARIABLE

**RF networks without the general distress variable.** At age 14, the network invariance test was not significant for the RF networks without the general distress variable (M = .15, p = .30; see Figure C.7), but the global network expected influence (EI) differed between the CA+ and the CA- RF networks ($EI_{CA+}$ = 3.21, $EI_{CA-}$ = 3.53, EI = 0.31, p = .03). More specifically, the global network expected influence was higher in the CA- than in the CA+ network. Those findings were only partially similar in the RF networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .12, p = .56; $EI_{CA+}$ = 3.54, $EI_{CA-}$ = 3.35, EI = 0.19, p = .16). At age 14 six interrelations differed between the CA+ and the CA- networks: namely friendship support and brooding (CA+: less positive; CA-:more positive), friendship support and expressive suppression (CA+: negative; CA-: positive), positive self-esteem and brooding (CA+: null; CA-: positive), positive self-esteem and expressive suppression (CA+: null; CA-: negative), brooding and aggression (CA+: null; CA-: positive), as well as reflection and distress tolerance (CA+: null; CA-: positive). At age 17, the interrelation between friendship support and positive self-esteem (CA+: positive, CA-: null) differed between the CA+ and the CA- network.

Interestingly, when we compared the RF networks for age 14 and age 17, we did find a global network structure differences for adolescents with (M = .22, p = .003; $EI_{14}$ = 3.21, $EI_{17}$ = 3.54, EI = 0.33, p = .001), but not for adolescents without a history of adversity (M = .17, p = .24; $EI_{14}$ = 3.53, $EI_{17}$ = 3.35, EI = 0.18, p = .22). In the CA+ network five RF interrelations changed from age 14 to age 17, namely the interrelation between friendship support and expressive suppression (from negative to positive), positive and negative self-esteem (from more to less positive), positive self-esteem and aggression (from null to positive), negative self-esteem and brooding (from more to less positive), and the interrelation between negative self-esteem and aggression (from less to more positive). In the CA- network, three RF interrelations changed from age 14 to age 17, namely the interrelation between friendship support and negative self-esteem (from positive to more positive), negative self-esteem and aggression (from positive to more positive), and the interrelation between brooding and reflection (from more to less positive).

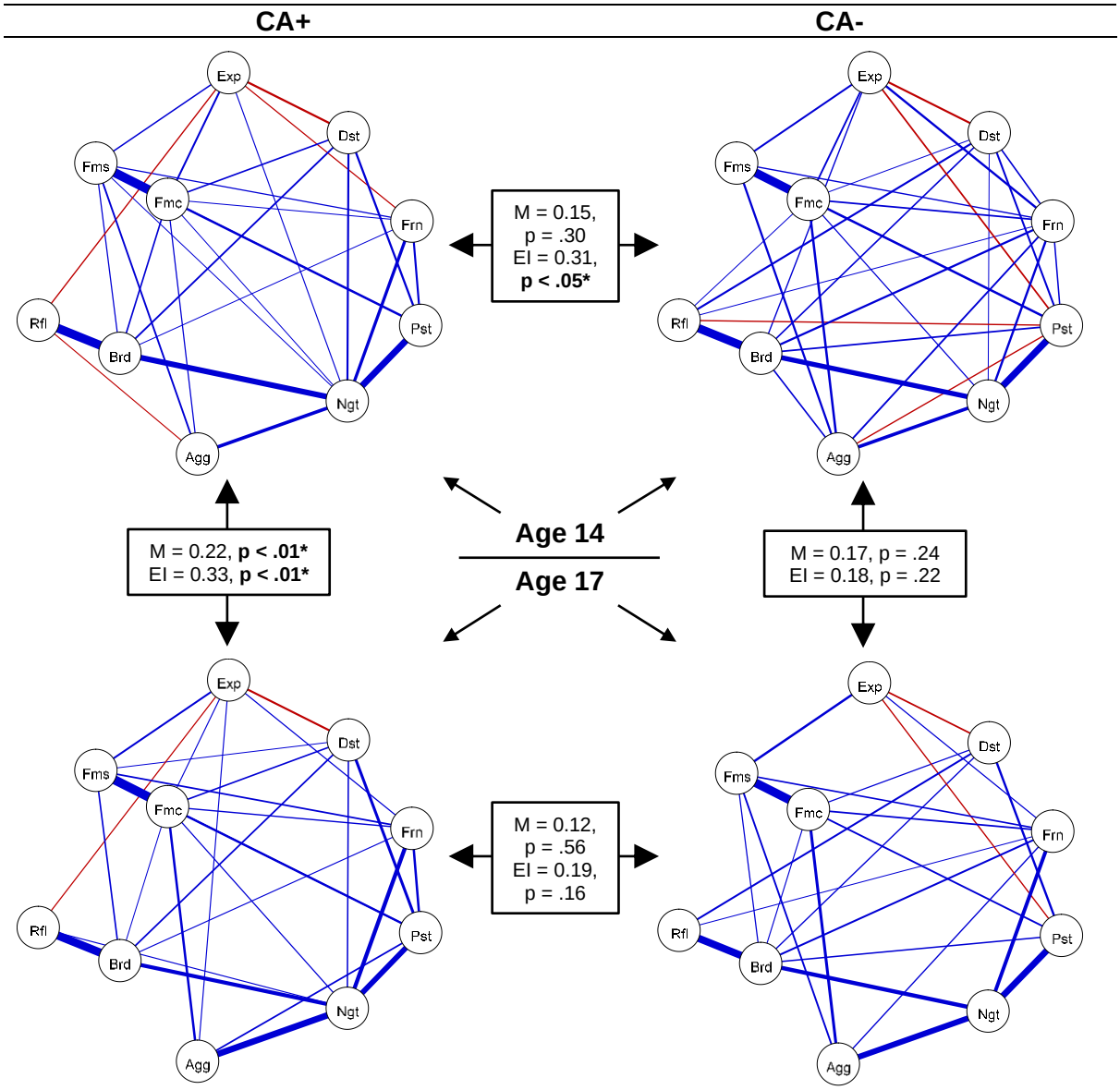



*Figure C.7.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for age 14 (upper panel) and age 17 (lower panel) without the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples). The above networks with faded interrelations can be found in Appendix C.14.

**RF networks with the general distress variable**. At age 14, the network invariance test was not significant for the RF networks with the general distress variable (M = .14, p = .47; see Figure C.8). However, the network expected influence was significantly higher in the CA- than in the CA+ network ($EI_{CA+}$ = 0.87, $EI_{CA-}$ = 1.43, EI = 0.56, p = .04). Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group at age 17 (M = .16, p = .34; $EI_{CA+}$ = 0.97, $EI_{CA-}$ = 1.16, EI = 0.19, p = .54). At age 14, four interrelation differed between the CA+ and the CA- network, namely the interrelations between friendship support and brooding (CA+: null, CA-: positive), friendship support and expressive suppression (CA+: negative, CA-: positive), positive self-esteem and expressive suppression (CA+: null, CA-: negative), as well as between reflection and distress tolerance (CA+: null, CA-: positive). At age 17 two interrelations differed between the CA+ and the CA- network, namely friendship support and positive self-esteem (CA+: positive, CA-: null), as well as aggression and general distress (CA+: more negative, CA-: less negative).

When we compared those networks for age 14 and age 17, the network invariance test was still significant (M = .20, p = .01), but the global network expected influence did no longer differ ($EI_{14}$ = 0.87, $EI_{17}$ = 0.97, EI = 0.11, p = .71) in the CA+ group. In the CA- group, neither of the two tests was significant (M = .12, p = .79; $EI_{14}$ = 1.43, $EI_{17}$ = 1.16, EI = 0.26, p = .36). In the CA+ network five individual RF interrelations changed from age 14 to age 17, namely the interrelations between friendship support and expressive suppression (from negative to positive), family cohesion and general distress (from more to less negative), negative self-esteem and brooding (from more to less positive), brooding and general distress (from more to less negative), as well as between aggression and general distress (from less to more negative). In contrast in the CA- network, only two RF interrelations changed from age 14 to age 17, namely the interrelation between negative self-esteem and general distress (from less to more negative), as well as the interrelation between brooding and general distress (from more to less negative).

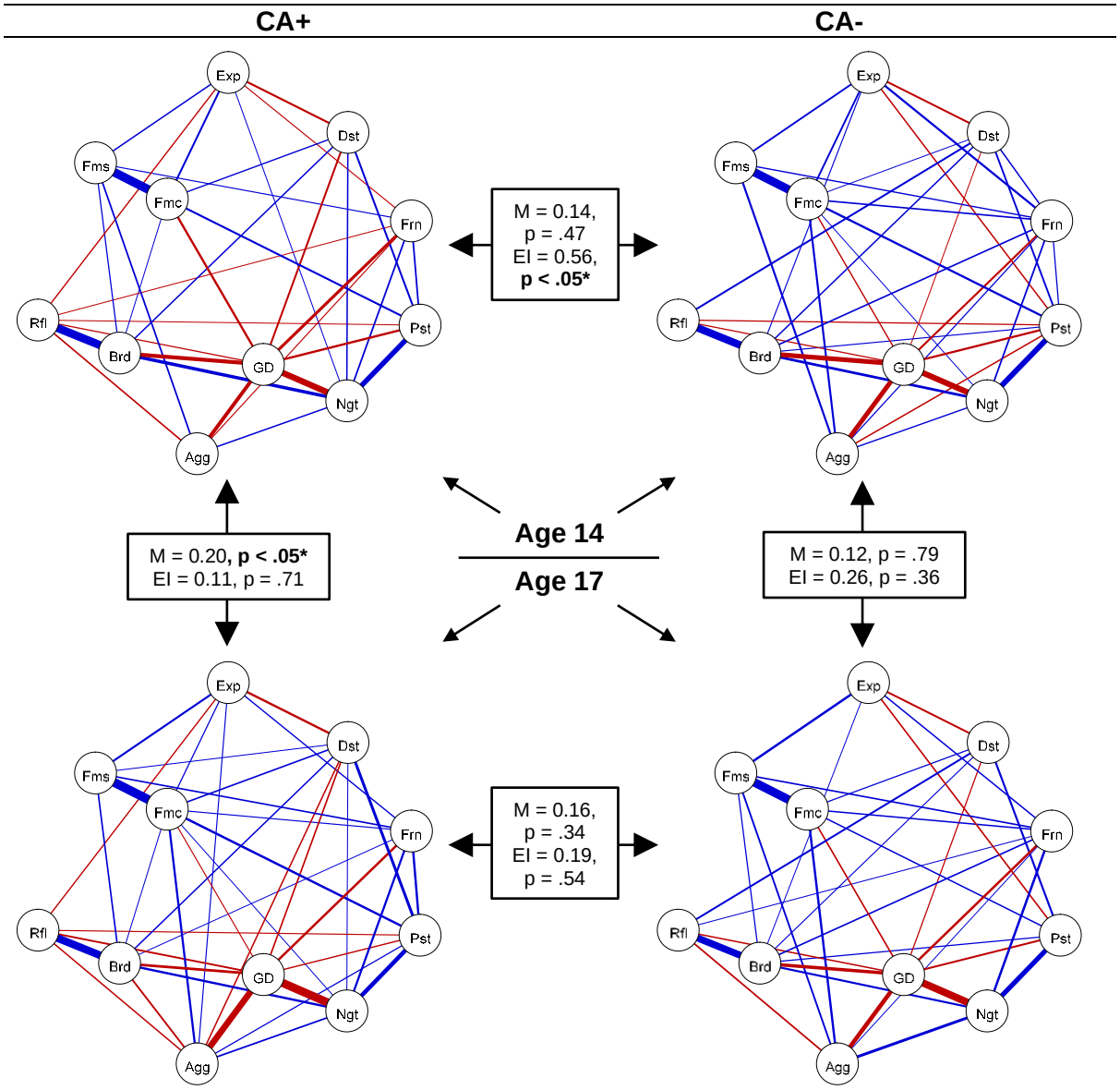



*Figure C.8.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for age 14 (upper panel) and age 17 (lower panel) with the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples). The above networks with faded interrelations can be found in Appendix C.14.

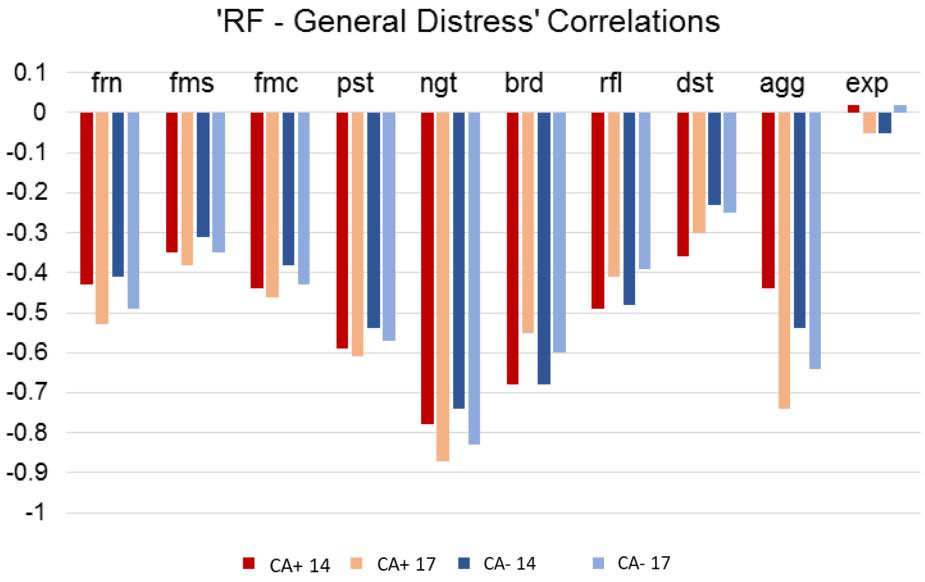
## APPENDIX C.8: CORRELATIONS AND REGULARIZED PARTIAL CORRELATIONS BETWEEN THE RESILIENCE FACTORS AND THE GENERAL DISTRESS FACTOR

As can be seen in Table C.6, all RFs were negatively correlated with general distress, except for expressive suppression for CA+ adolescents at age 14 and CA- adolescents at age 17, as it then was positively correlated with general distress. Regularized partial correlations of the RFs, which we used for the network models, were also mostly negative for the relationships between the RFs and general distress. Yet, expressive suppression and family support were in both groups no longer related with general distress, neither at age 14 nor at 17. Thus, the overall results pattern was still similar, showing that even after the correction for all other RFs, most RFs were clearly negatively associated with general distress. As can be seen in Figure C.9, negative self-esteem, positive self-esteem, brooding and aggression seemed to be most strongly correlated with general distress. However, in terms of partial correlations, positive self-esteem no longer appeared to be among those factors with the highest interrelations with general distress.

Table C.6
*Correlations and regularized partial correlations between the RFs and the general distress variable*

| CA | frn | fms | fmc | pst | ngt | brd | rfl | dst | agg | exp |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | *Correlations* | | | | | |
| yes: age 14 | -0.43 | -0.35 | -0.44 | -0.59 | -0.78 | -0.68 | -0.49 | -0.36 | -0.44 | 0.02 |
| yes: age 17 | -0.53 | -0.38 | -0.46 | -0.61 | -0.87 | -0.55 | -0.41 | -0.30 | -0.74 | -0.05 |
| no: age 14 | -0.41 | -0.31 | -0.38 | -0.54 | -0.74 | -0.68 | -0.48 | -0.23 | -0.54 | -0.05 |
| no: age 17 | -0.49 | -0.35 | -0.43 | -0.57 | -0.83 | -0.60 | -0.39 | -0.25 | -0.64 | 0.02 |
| | | | | | *Regularized Partical Correlations* | | | | | |
| yes: age 14 | -0.17 | 0.00 | -0.12 | -0.11 | -0.42 | -0.25 | -0.03 | -0.08 | -0.22 | 0.00 |
| yes: age 17 | -0.13 | 0.00 | -0.03 | -0.04 | -0.58 | -0.14 | -0.08 | -0.06 | -0.42 | 0.00 |
| no: age 14 | -0.10 | 0.00 | -0.04 | -0.11 | -0.39 | -0.30 | -0.03 | -0.02 | -0.29 | 0.00 |
| no: age 17 | -0.12 | 0.00 | -0.06 | -0.09 | -0.51 | -0.20 | -0.05 | -0.04 | -0.26 | 0.00 |

*Note.* CA = Childhood adversity (yes: *n* = 631, no: *n* = 499). Frn = friend support, fms = family support, fmc = family cohesion, pst = positive self-esteem, ngt = negative self-esteem, brd = brooding, rfl = reflection, dst = distress tolerance, agg = aggression, exp = expressive suppression.



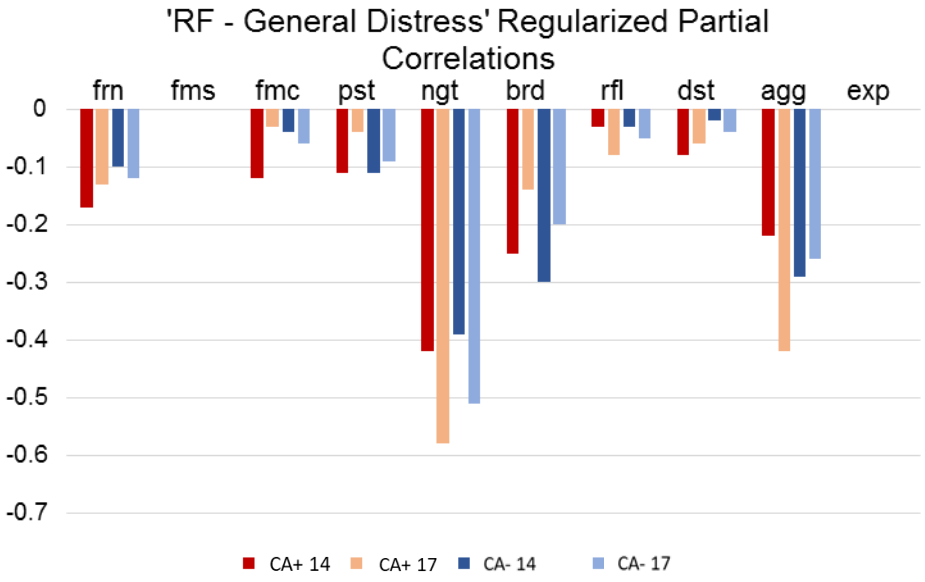




*Figure C.9.* Visualizing the correlations and regularized partial correlations between RFs and general distress, for CA+ (*n* = 631) and CA- (*n* = 499) adolescents at age 14 and age 17.

## APPENDIX C.9: THE STABILITY OF THE EXPECTED INFLUENCE (EI) COEFFICIENTS AND THE ACCURACY OF THE RESILIENCE FACTOR INTERRELATIONS AND 'RESILIENCE FACTOR - GENERAL DISTRESS' INTERRELATIONS

To test the stability of the *expected influence (EI)* coefficients we applied a subset bootstrap (2000 bootstraps) to identify the maximum sample percentage that can be dropped to reveal (with a 95% chance) a relationship of ≥0.7 between the subset and the original EI coefficients. The analyses showed that at both age 14 and 17 EI coefficients were sufficiently stable, as more than 50 percent of the sample could be dropped (see Table C.7).

To test the accuracy of the network models we bootstrapped the 'RF-RF' and 'RF-general distress' interrelations (2000 bootstraps) and investigated the bootstrap confidence intervals (CIs). Overall, the CIs had an acceptable width, which indicates that the estimated models have an appropriate interrelation accuracy. At age 14, family cohesion and family support were most strongly interrelated, followed next by the brooding and reflective rumination interrelation, and then by the positive and negative self-esteem interrelation, for both the CA+ and the CA- group (see Figure C.10 and Figure C.11). In the models without general distress, negative self-esteem and aggression were additionally very highly interrelated, at age 17 (see Figure C.10). In the models with general distress, negative self-esteem and general distress were also strongly interrelated (see Figure C.11).

Table C.7
*Expected influence (EI) stability*

| CA | Age | $MDP_{EI}$ | Case range for $MDP_{EI}$ | $MDP_{EI}$ | Case range for $MDP_{EI}$ |
|---|---|---|---|---|---|
| | | *Without general distress* | | *With general distress* | |
| Yes | 14 | 0.750 | (caseMin = 0.721, caseMax = 1) | 0.750 | (caseMin = 0.721, caseMax = 1) |
| No | 14 | 0.749 | (caseMin = 0.721, caseMax = 1) | 0.749 | (caseMin = 0.721, caseMax = 1) |
| Yes | 17 | 0.750 | (caseMin = 0.721, caseMax = 1) | 0.750 | (caseMin = 0.721, caseMax = 1) |
| No | 17 | 0.749 | (caseMin = 0.721, caseMax = 1) | 0.749 | (caseMin = 0.721, caseMax = 1) |

*Note.* CA = childhood adversity. MDP = Maximum drop proportion.

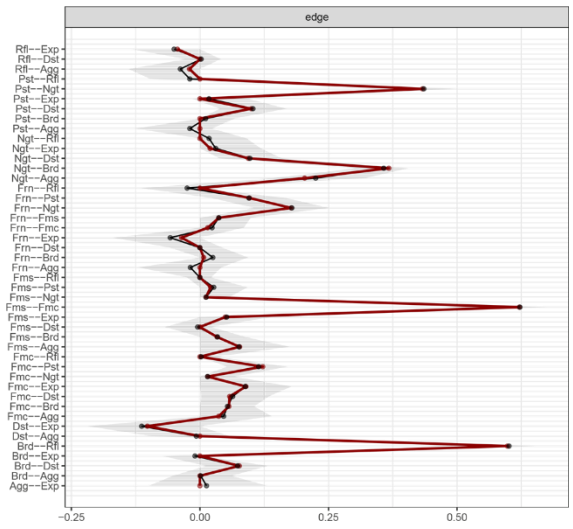
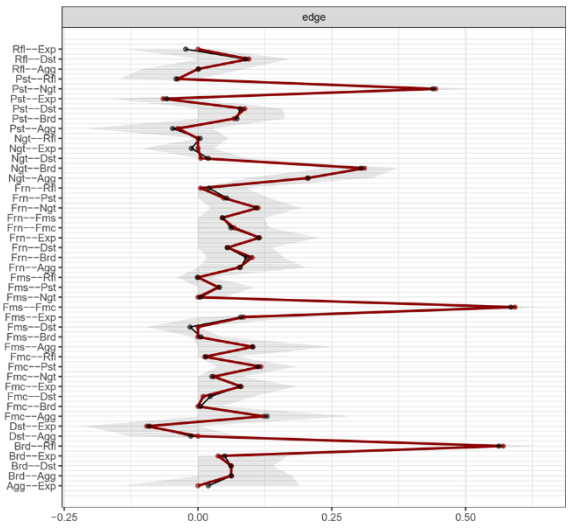
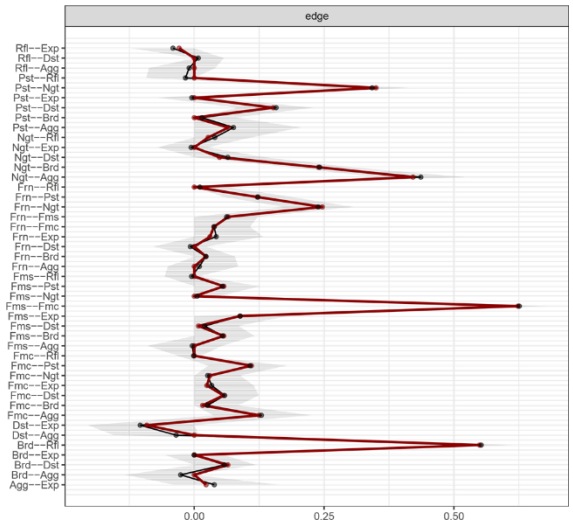
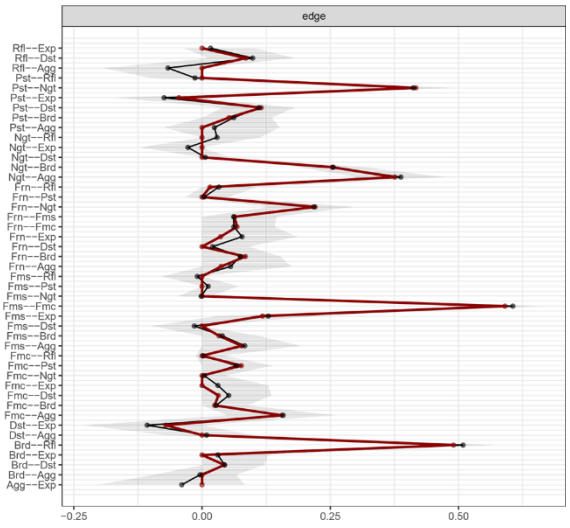
| CA+ | CA- |
|---|---|
| *Age 14, without general distress* | |
|  |  |
| *Age 17, without general distress* | |
|  |  |

*Figure C.10.* Interrelation accuracy plots for CA+ (*n* = 631) and CA- (*n* = 499) networks without the general distress variable, for both age 14 and age 17. The plots depict the sample RF interrelations (i.e. edge weights) which are represented by the red dots, the means of the bootstrapped RF interrelations (i.e. edge weights) which are represented by the black dots, and the belonging bootstrap confidence intervals (CIs) which indicate the RF interrelation accuracy.

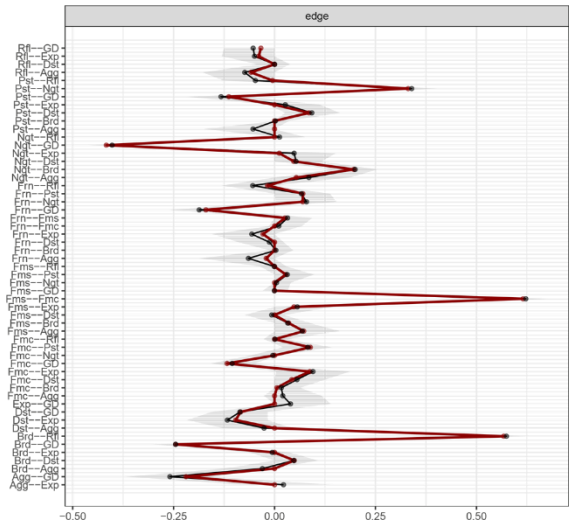
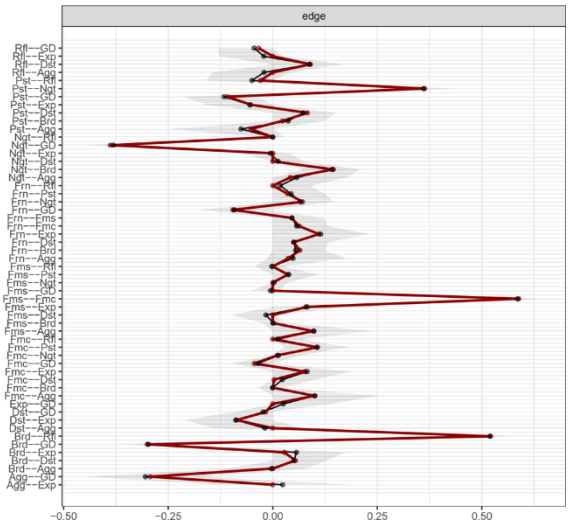
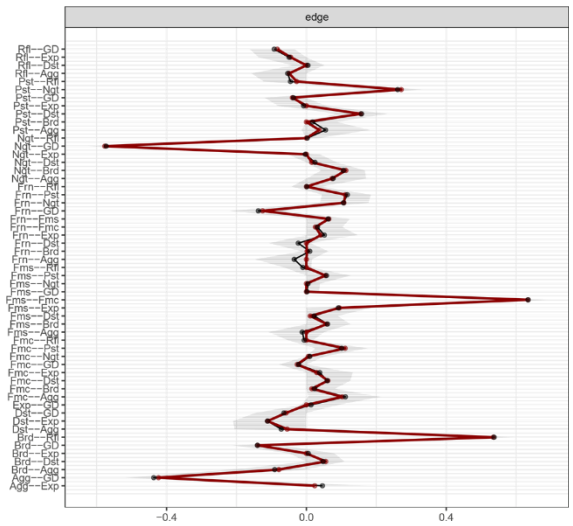
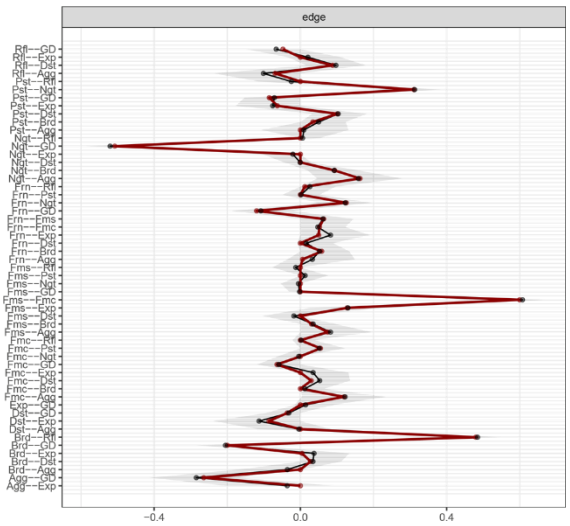
| CA+ | CA- |
|---|---|
| *Age 14, with general distress* | |
|  |  |
| *Age 17, with general distress* | |
|  |  |

*Figure C.11.* Interrelation accuracy plots for CA+ (*n* = 631) and CA- (*n* = 499) networks with the general distress variable, for both age 14 and age 17. The plots depict the sample 'RF-RF' and 'RF-general distress' interrelations (i.e. edge weights) which are represented by the red dots, the means of the bootstrapped interrelations (i.e. edge weights) which are represented by the black dots, and the belonging bootstrap confidence intervals (CIs) which indicate the 'RF-RF' and 'RF-general distress' interrelation accuracy.

## APPENDIX C.10: EXPECTED INFLUENCE (EI) FOR RESILIENCE FACTORS IN NETWORKS CORRECTED FOR GENERAL DISTRESS

The connectivity analyses for expected influence (EI) coefficients showed that the family, ruminative brooding and negative self-esteem RFs had the highest coefficients, in both groups and at both time points (see Table C.8). For both group, expressive suppression had the lowest EI coefficient, at both time points. We did not detect any particular age or group patterns (see Figure C.12).

Table C.8
*Expected influence (EI) for networks corrected for general distress*

| CA | frn | fms | fmc | pst | ngt | brd | rfl | dst | agg | exp |
|---|---|---|---|---|---|---|---|---|---|---|
| yes: age 14 | 0.10 | 0.83 | 0.84 | 0.60 | 0.71 | 0.85 | 0.45 | 0.13 | 0.05 | -0.02 |
| yes: age 17 | 0.35 | 0.92 | 0.99 | 0.71 | 0.59 | 0.70 | 0.41 | 0.13 | 0.06 | 0.03 |
| no: age 14 | 0.48 | 0.85 | 0.95 | 0.51 | 0.63 | 0.84 | 0.58 | 0.19 | 0.22 | 0.16 |
| no: age 17 | 0.37 | 0.90 | 0.86 | 0.44 | 0.69 | 0.73 | 0.51 | 0.16 | 0.29 | 0.03 |

*Note.* CA = Childhood adversity (yes: *n* = 631, no: *n* = 499). Frn = friend support, fms = family support, fmc = family cohesion, pst = positive self-esteem, ngt = negative self-esteem, brd = brooding, rfl = reflection, dst = distress tolerance, agg = aggression, exp = expressive suppression.

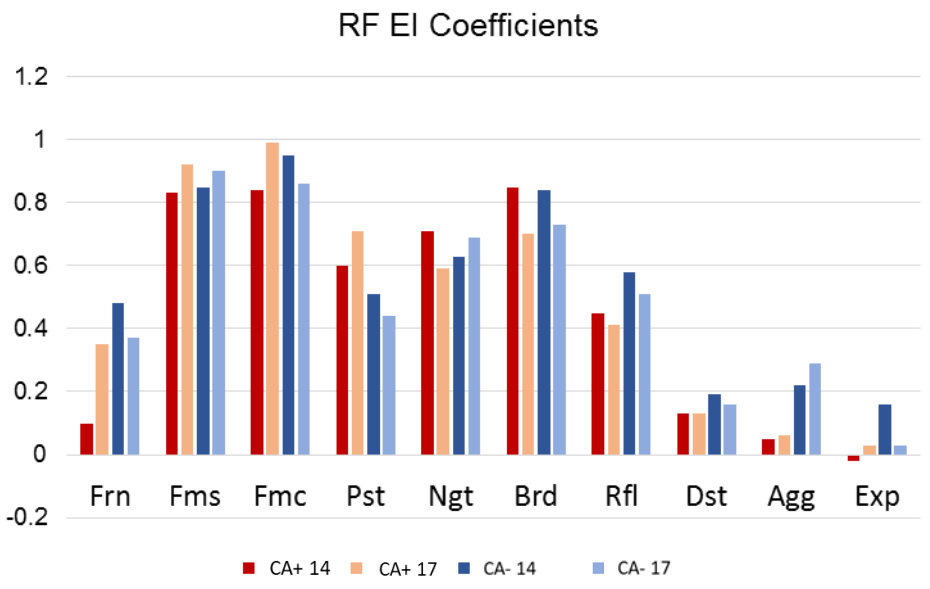



*Figure C.12.* Visualizing expected influence (EI) coefficients for CA+ (*n* = 631) and CA- (*n* = 499) networks corrected for the general distress variable, for both age 14 and age 17. Frn = friend support, fms = family support, fmc = family cohesion, pst = positive self-esteem, ngt = negative self-esteem, brd = brooding, rfl = reflection, dst = distress tolerance, agg = aggression, exp = expressive suppression.

## APPENDIX C.11: NETWORK ANALYSIS RESULTS CONDUCTED WITH FACTOR SCORES DERIVED FROM THE CONFIGURAL LCFA MODELS

The following three figures depict CA+ and CA- networks with factor scores derived from the configural CFA models. The models were estimated separately for age 14 and 17, as well as (1) once without the general distress variable, (2) once with the general distress variable, and (3) once corrected for the general distress variable. At age 14, the network invariance test was not significant for the networks without the general distress variable (M = .16, p = .24; see Figure C.13), but the global network expected influence differed between the CA+ and the CA- RF networks ($EI_{CA+}$ = 3.22, $EI_{CA-}$ = 3.54, EI = 0.32, p = .03). More specifically, the RFs in the CA+ network were less positively interrelated. Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .12, p = .57; $EI_{CA+}$ = 3.55, $EI_{CA-}$ = 3.34, EI = 0.21, p = .13). When we compared the RF networks for age 14 and age 17, the two CA+ networks were not invariant over time, in other words, they did vary over time (M = .24, p < .001). Moreover, the RFs in the age 14 network were less positively interrelated than in the age 17 network ($EI_{14}$ = 3.22, $EI_{17}$ = 3.55, EI = 0.33, p = .001). The age 14 and age 17 CA- networks did however not differ with regard to their global network structure (M = .17, p = .23; $EI_{14}$ = 3.54, $EI_{17}$ = 3.34, EI = 0.20, p = .17).

For the networks with the general distress variable, the network invariance test (M = .17, p = .15; see Figure C.14) was not significant at age 14. However, the global network expected influence differed between the CA+ and the CA- networks ($EI_{CA+}$ = 0.69, $EI_{CA-}$ = 1.39, EI = 0.70, p = .01). Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .17, p = .26; $EI_{CA+}$ = 0.91, $EI_{CA-}$ = 1.17, EI = 0.26, p = .40). When we compared the CA+ networks for age 14 and age 17, the network invariance test was significant (M = .19, p = .03), but the network expected influence did not differ ($EI_{14}$ = 0.69, $EI_{17}$ = 0.91, EI = 0.22, p = .49). The age 14 and age 17 CA- networks did again not differ with regard to their global network structure (M = .12, p = .84; $EI_{14}$ = 1.39, $EI_{17}$ = 1.17, EI = 0.22, p = .42).

For the networks corrected for the general distress variable, the network invariance test (M = .17, p = .13; see Figure C.15) was not significant, at age 14, but the global network expected influence differed between the CA+ and the CA- networks ($EI_{CA+}$ = 2.15, $EI_{CA-}$ = 2.69, EI = 0.54, p = .005). Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .11, p = .86; $EI_{CA+}$ = 2.40, $EI_{CA-}$ = 2.49, EI = 0.09, p = .68). When we compared the networks for age 14 and age 17, we did not find any significant global network structure differences; neither for adolescents with (M = .10, p = .78; $EI_{14}$ = 2.15, $EI_{17}$ = 2.40, EI = 0.26, p = .21) nor for adolescents without a history of adversity (M = .12, p = .81; $EI_{14}$ = 2.69, $EI_{17}$ = 2.49, EI = 0.20, p = .30).

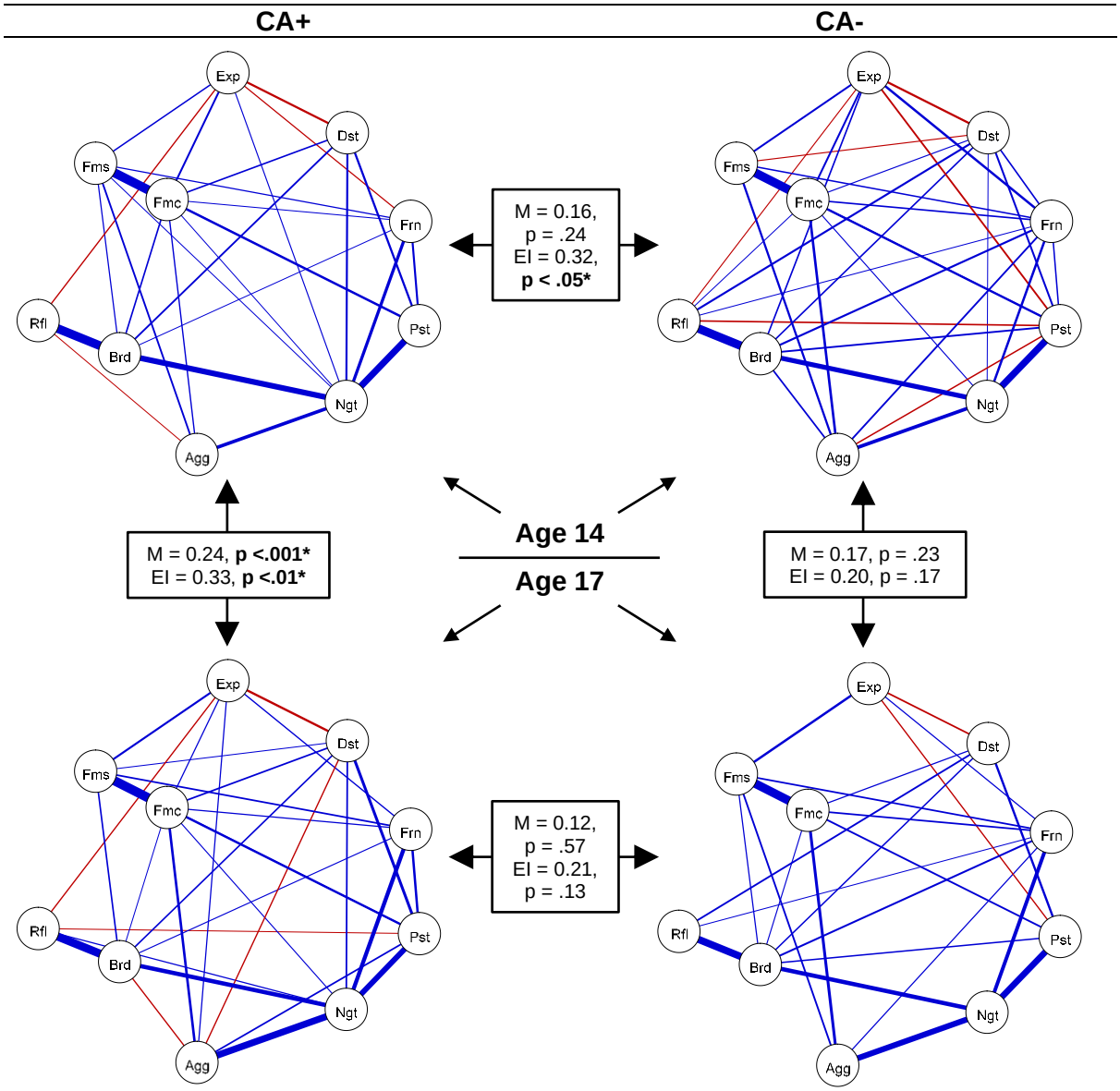



*Figure C.13.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks with configural factor scores for age 14 (upper panel) and age 17 (lower panel) without the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

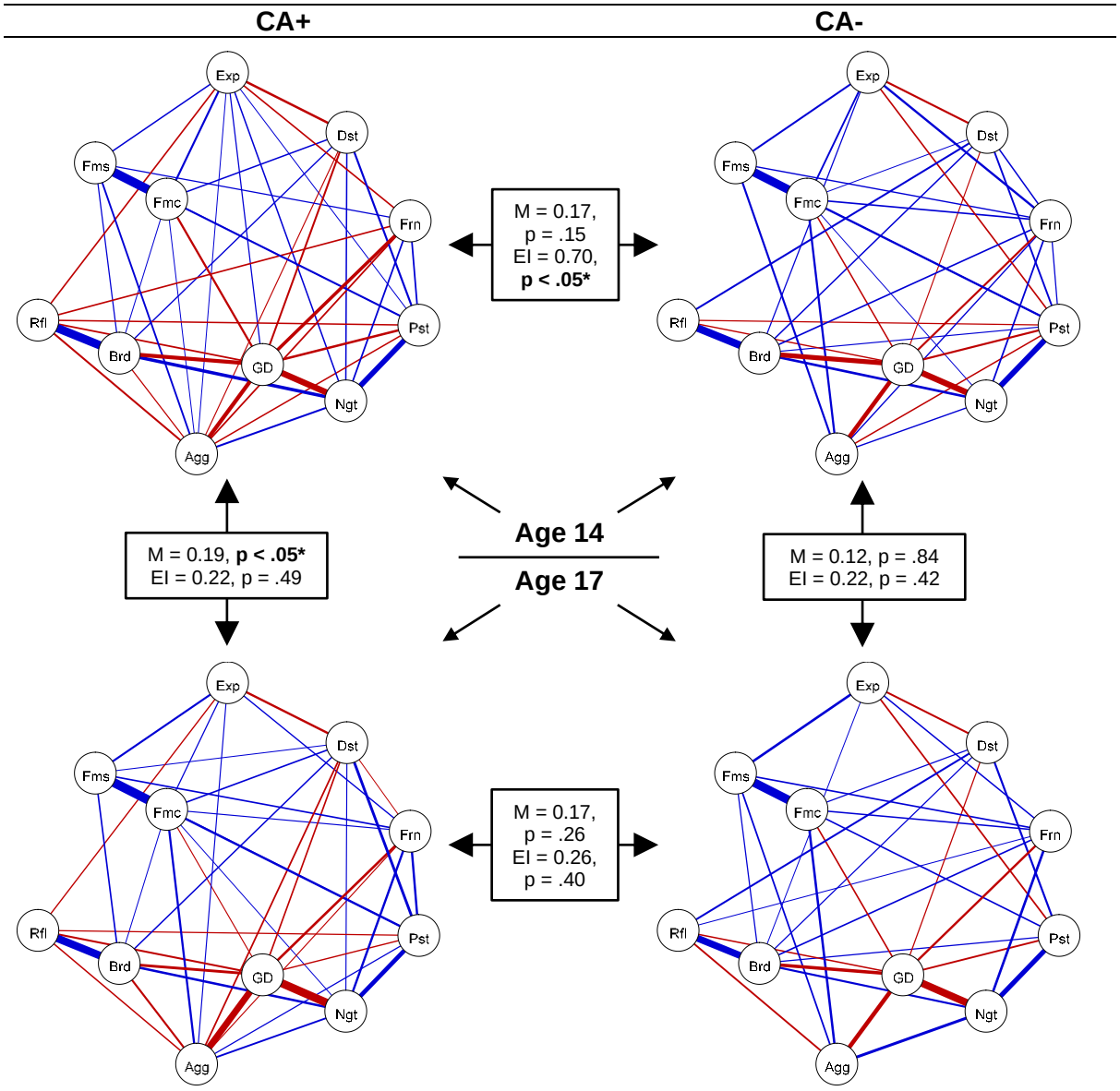



*Figure C.14.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks with configural factor scores for age 14 (upper panel) and age 17 (lower panel) with the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

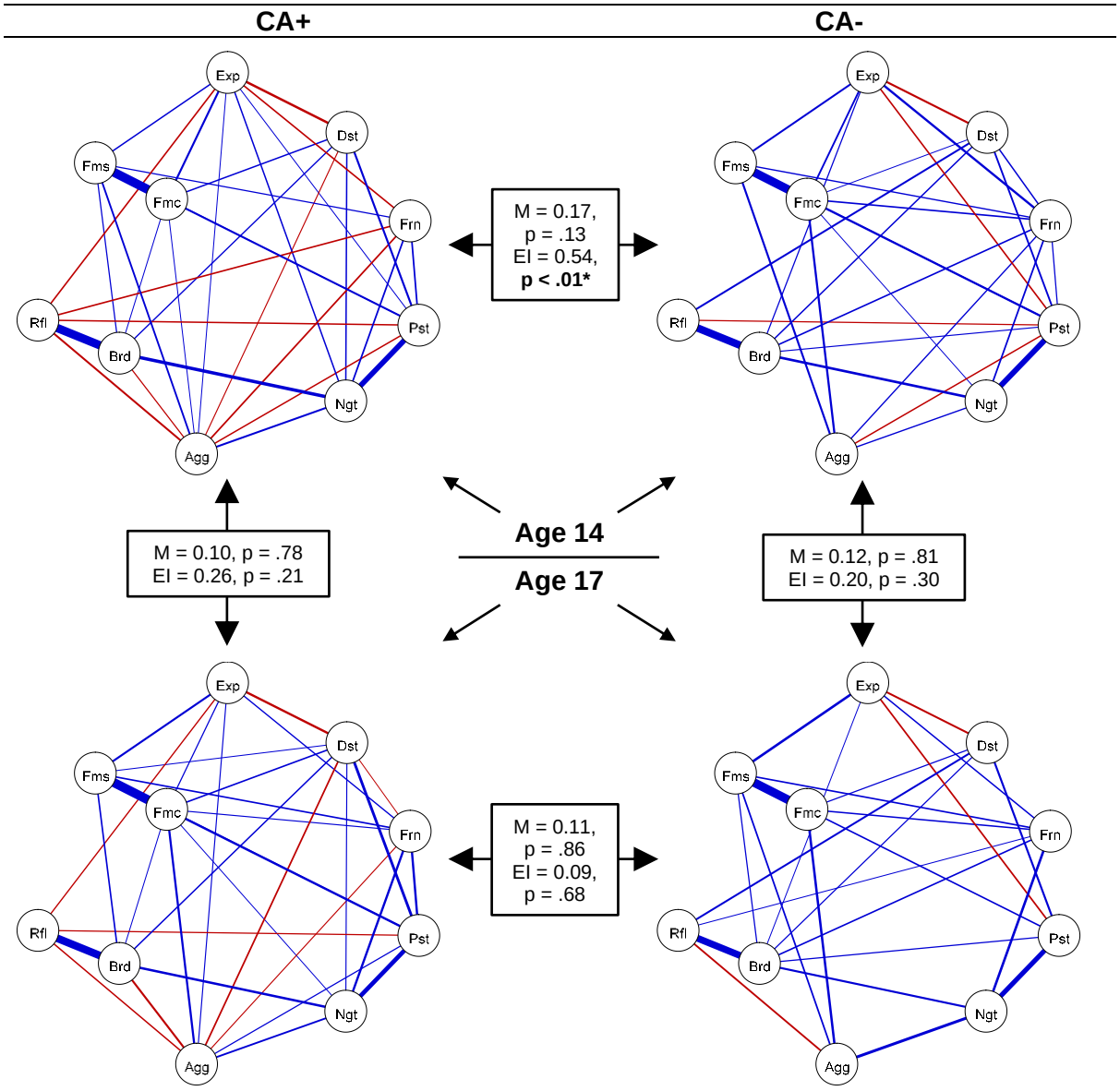



*Figure C.15.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for configural factor scores for age 14 (upper panel) and age 17 (lower panel) corrected for the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

## APPENDIX C.12: NETWORK ANALYSIS RESULTS CONDUCTED WITH SUM SCORES

The following three figures depict CA+ and CA- networks with sum scores. The models were estimated separately for age 14 and 17, as well as (1) once without the general distress variable, (2) once with the general distress variable, and (3) once corrected for the general distress variable. At age 14, the network invariance test was not significant for the networks without the general distress variable (M = .12, p = .58; see Figure C.16), and the global network expected influence differed marginally between the CA+ and the CA- RF networks ($EI_{CA+}$ = 3.01, $EI_{CA-}$ = 3.31, EI = 0.31, p = .08). More specifically, the RFs in the CA+ network were less positively interrelated. Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .13, p = .48; $EI_{CA+}$ = 3.43, $EI_{CA-}$ = 3.33, EI = 0.10, p = .49). When we compared the RF networks for age 14 and age 17, the two CA+ networks were not invariant over time, in other words, they did vary over time (M = .24, p = .001). Moreover, the RFs in the age 14 network were less positively interrelated than in the age 17 network ($EI_{14}$ = 3.01, $EI_{17}$ = 3.43, EI = 0.42, p < .001). The two CA- networks were not invariant over time (M = .22, p = .03), but did not differ in expected influence ($EI_{14}$ = 3.31, $EI_{17}$ = 3.33, EI = 0.02, p = .93).

For the networks with the general distress variable, the network invariance test (M = .13, p = .35; see Figure C.17) was not significant at age 14. However, the global network expected influence differed between the CA+ and the CA- networks ($EI_{CA+}$ = 0.67, $EI_{CA-}$ = 1.20, EI = 0.53, p = .05). Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .13, p = .60; $EI_{CA+}$ = 1.22, $EI_{CA-}$ = 1.27, EI = 0.05, p = .84). When we compared the CA+ networks for age 14 and age 17, the network invariance test was significant (M = .19, p = .02), and the RFs in the age 14 network were less positively interrelated than in the age 17 network ($EI_{14}$ = 0.67, $EI_{17}$ = 1.22, EI = 0.55, p = .04). The age 14 and age 17 CA- networks were again not invariant (M = .25, p = 0.009), but did not differ with regard to the expected influence ($EI_{14}$ = 1.20, $EI_{17}$ = 1.27, EI = 0.07, p = .80).

For the networks corrected for the general distress variable, the network invariance test was not significant at age 14 (M = .13, p = .31; see Figure C.18), but the global network expected influence differed between the CA+ and the CA- networks ($EI_{CA+}$ = 2.09, $EI_{CA-}$ = 2.49, EI = 0.41, p = .04). Those findings were only partially similar in the networks for age 17, as neither of the two tests revealed significant differences between the CA+ and the CA- group (M = .13, p = .59; $EI_{CA+}$ = 2.51, $EI_{CA-}$ = 2.53, EI = 0.02, p = .90). When we compared the CA+ networks for age 14 and age 17, the network invariance test was not significant (M = .15, p = .12), but the RFs in the age 14 network were less positively interrelated than in the age 17 network ($EI_{14}$ = 2.09, $EI_{17}$ = 2.51, EI = 0.42, p = .02). The age 14 and age 17 CA- networks were invariant (M = .10, p = 0.90), and did not differ with regard to the expected influence ($EI_{14}$ = 2.49, $EI_{17}$ = 2.53, EI = 0.04, p = .85).

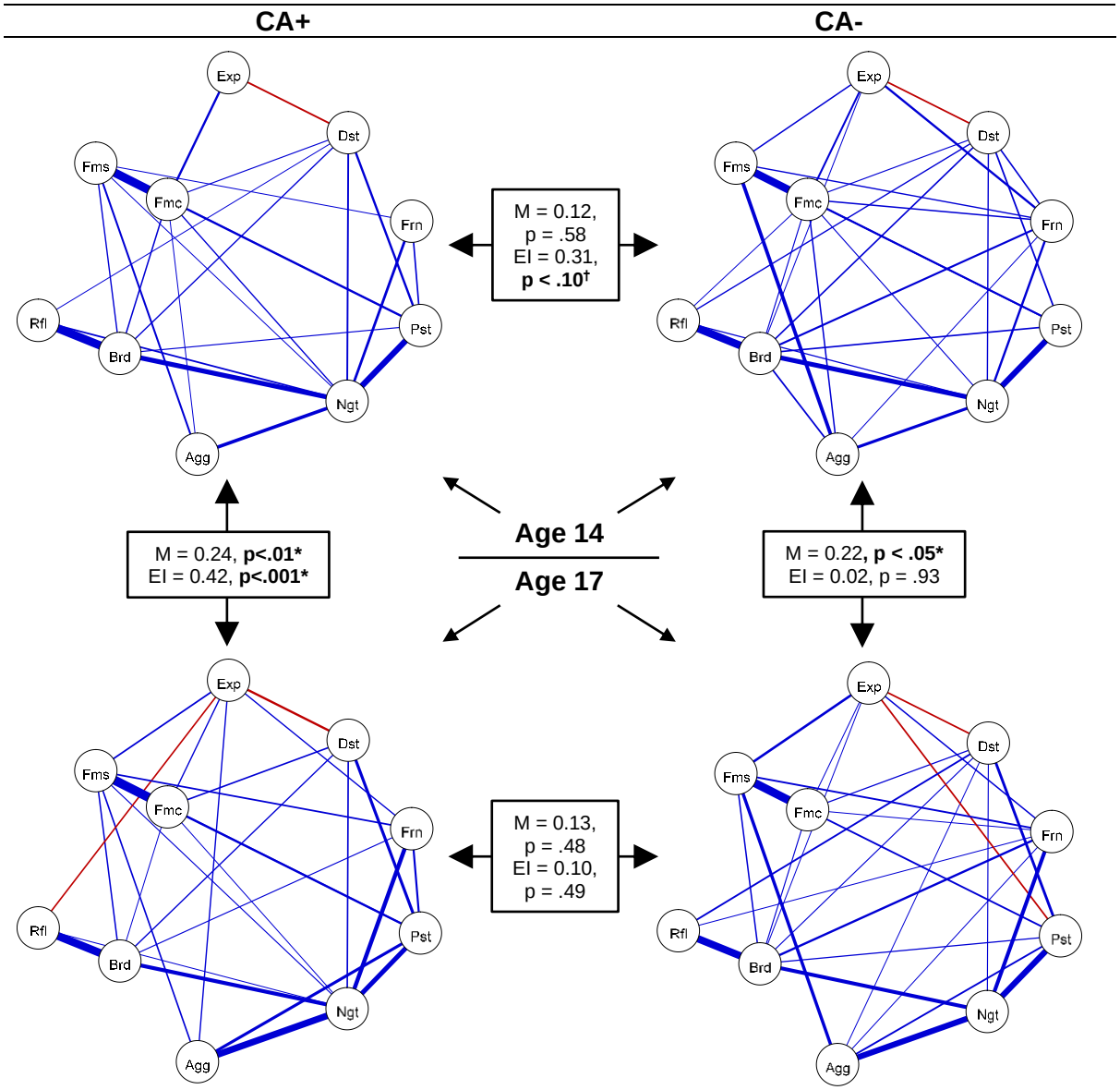



*Figure C.16.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks with sum scores for age 14 (upper panel) and age 17 (lower panel) without the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

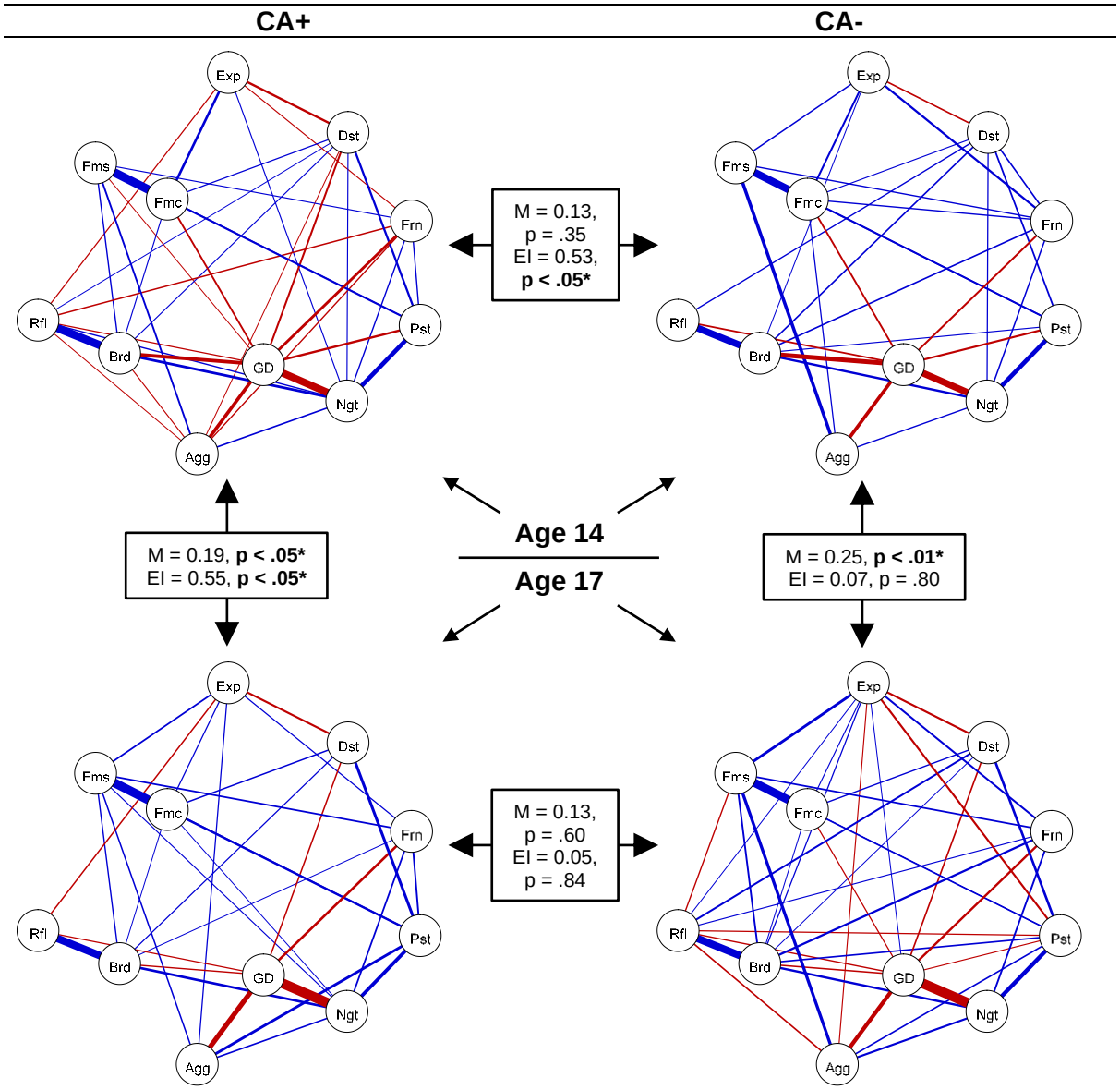



*Figure C.17.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks with sum scores for age 14 (upper panel) and age 17 (lower panel) with the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

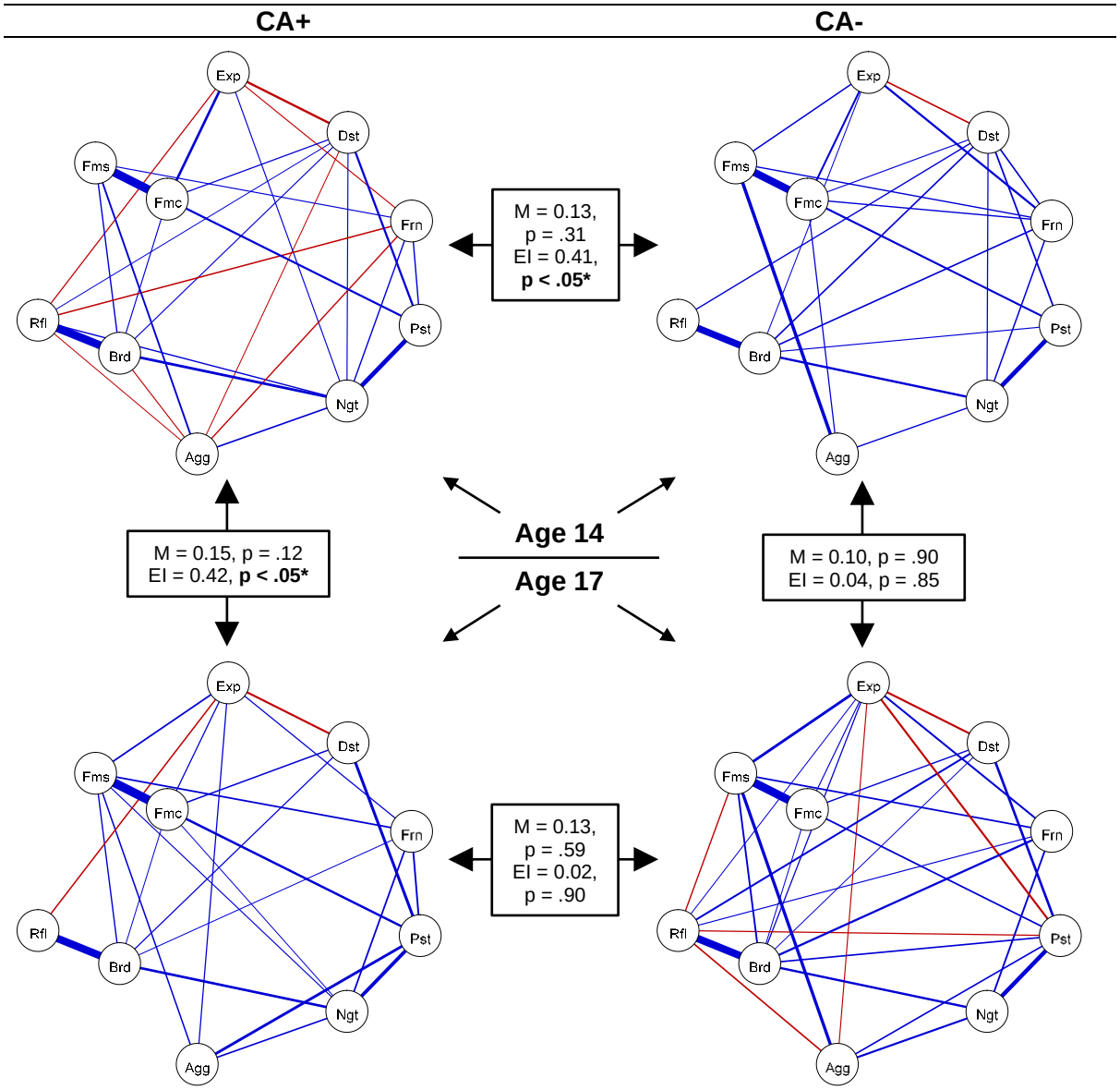



*Figure C.18.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks for sum scores for age 14 (upper panel) and age 17 (lower panel) corrected for the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

## APPENDIX C.13: SIGNIFICANT RESILIENCE FACTOR INTERRELATION DIFFERENCES (A) BETWEEN THE CA+ (N = 631) AND THE CA- (N = 499) NETWORKS, AS WELL AS (B) BETWEEN AGE 14 AND AGE 17 NETWORKS

*Significant RF-RF interrelation differences between the CA+ (n = 631) and the CA- (n = 499) networks*

| RF1 | RF2 | interrelation sign in the CA+ network | interrelation sign in the CA- network | E | *p* |
|---|---|---|---|---|---|
| | | Age 14 | | | |
| friendship support | brooding | null | positive | 0.07 | .014 |
| friendship support | expressive suppression | negative | positive | 0.14 | .007 |
| positive self-esteem | expressive suppression | null | negative | 0.05 | .046 |
| reflection | distress tolerance | null | positive | 0.09 | .015 |
| | | Age 17 | | | |
| friendship support | positive self-esteem | positive | null | 0.11 | .031 |

*Note.* RF = Resilience factor. CA = childhood adversity. E = RF-RF interrelation difference (i.e. edge difference).

*Significant RF-RF interrelation differences between age 14 and age 17 networks*

| RF1 | RF2 | interrelation sign in the age 14 network | interrelation sign in the age 17 network | E | *p* |
|---|---|---|---|---|---|
| | | CA+ Networks | | | |
| friendship support | expressive suppression | negative | positive | 0.07 | .031 |
| negative self-esteem | brooding | more positive | less positive | 0.08 | .019 |
| | | CA- Networks | | | |
| - | - | - | - | - | - |

*Note.* RF = Resilience factor. CA = childhood adversity. E = RF-RF interrelation difference (i.e. edge difference).

## APPENDIX C.14: NETWORK MODELS PRESENTED IN CHAPTER 4 AND IN APPENDIX C.7 WITH FADED INTERRELATIONS

The following three figures depict CA+ and CA- networks with faded interrelations, for both age 14 and age 17, for (1) the networks without the general distress variable, (2) the networks with the general distress variable, and (3) the networks corrected for the general distress variable.

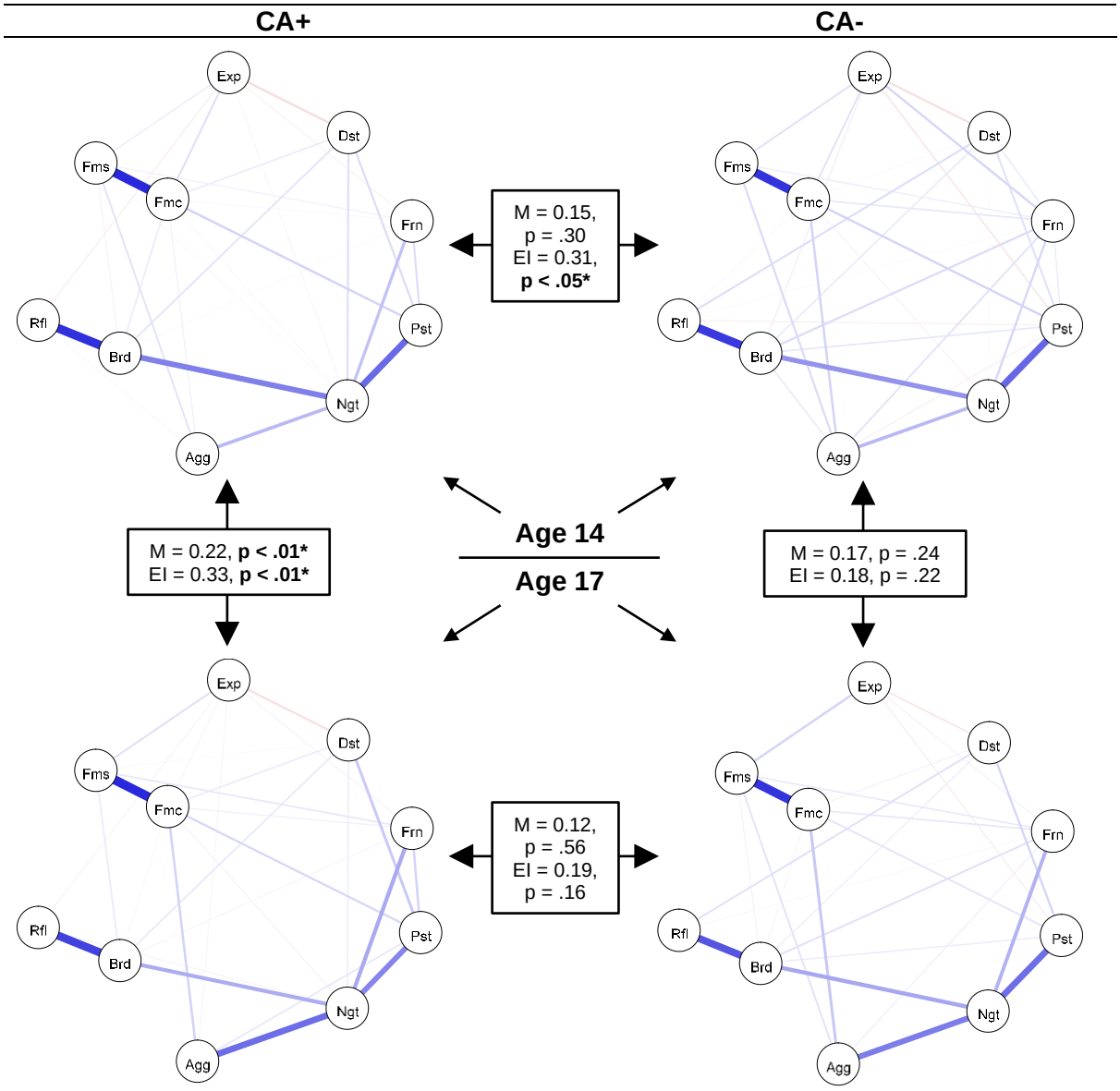



*Figure C.19.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks with faded interrelations for age 14 (upper panel) and age 17 (lower panel) without the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

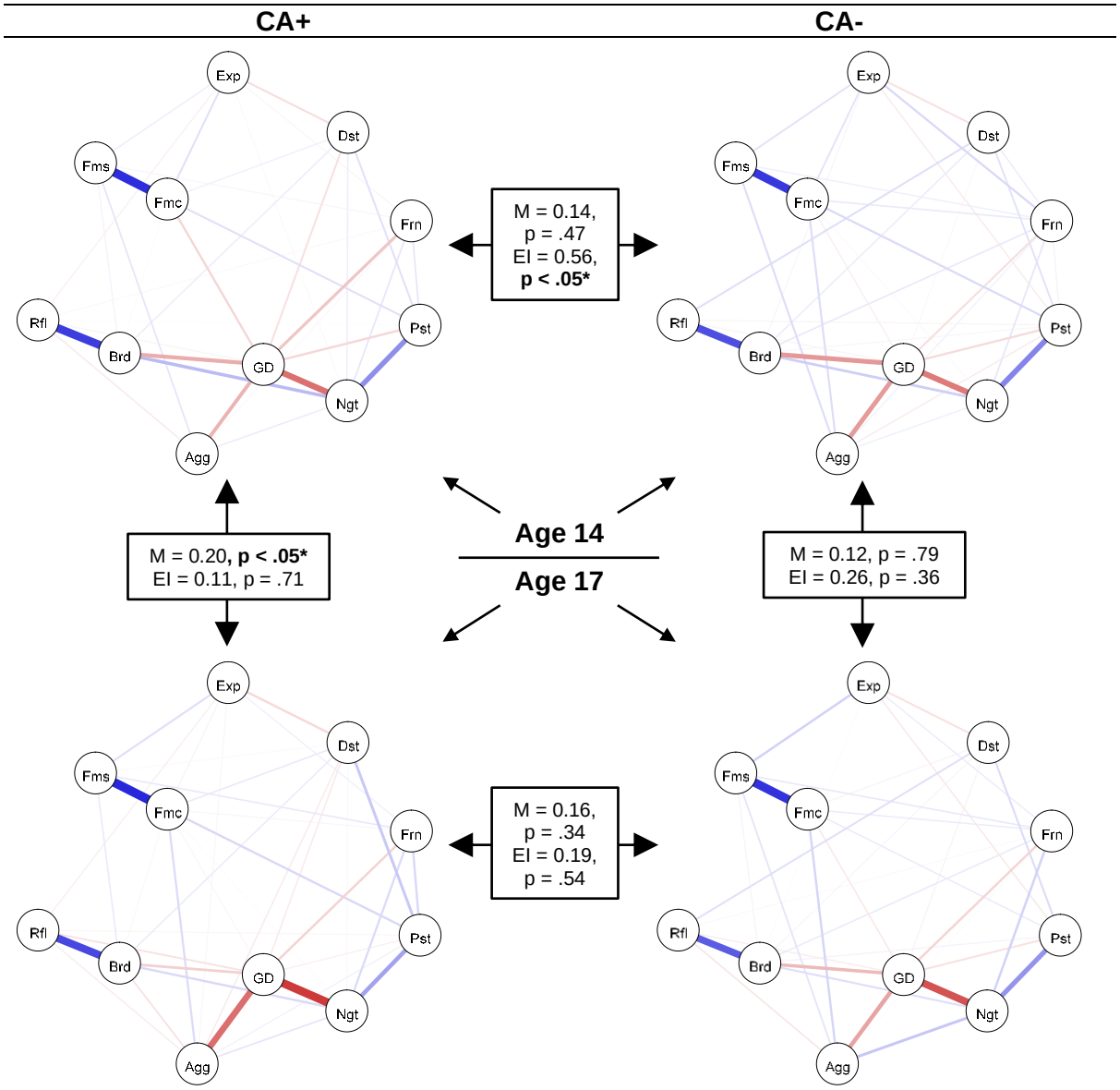



*Figure C.20.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks with faded interrelations for age 14 (upper panel) and age 17 (lower panel) with the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

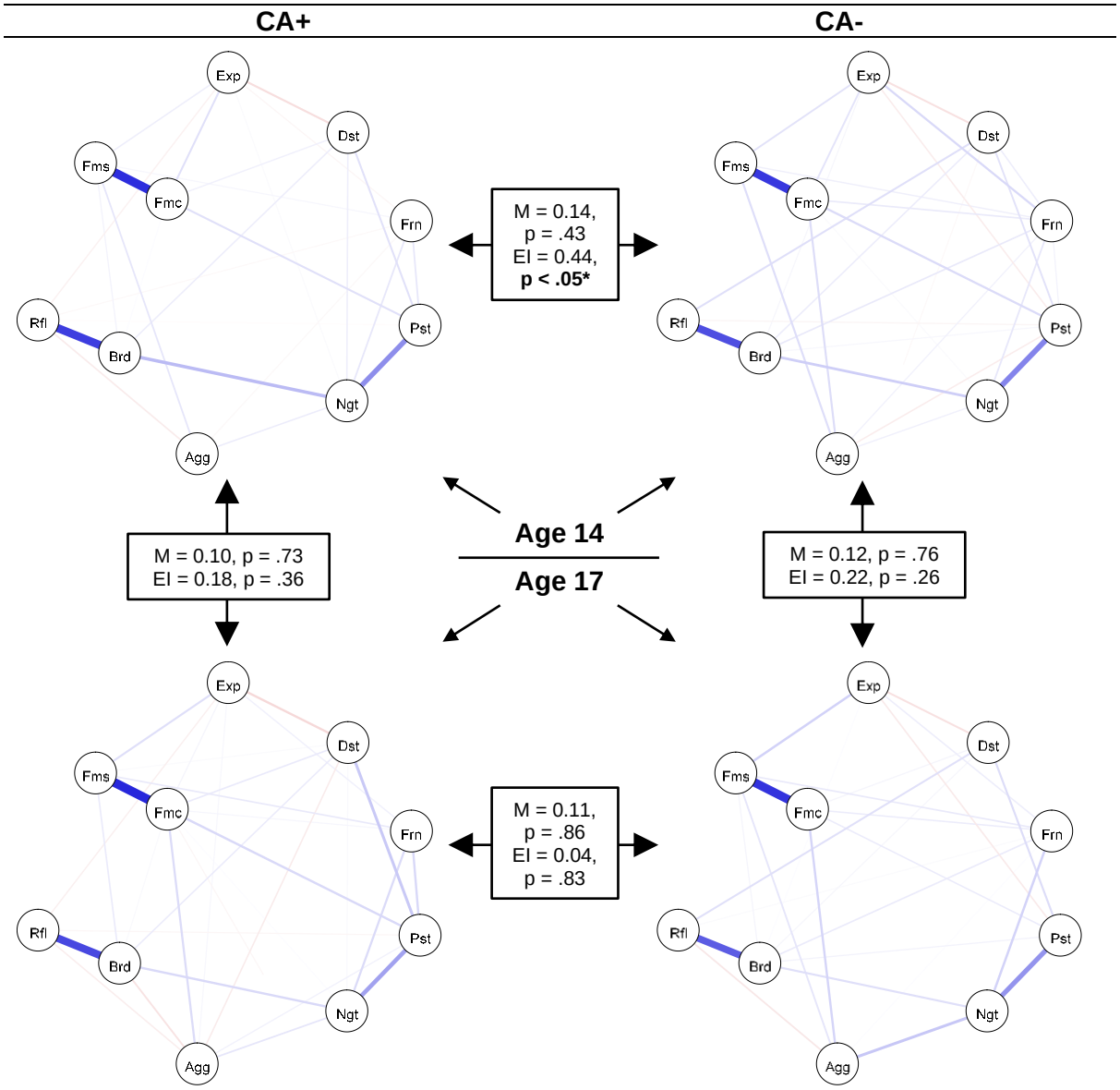



*Figure C.21.* CA+ (*n* = 631) and CA- (*n* = 499) resilience factor networks with faded interrelations for age 14 (upper panel) and age 17 (lower panel) corrected for the general distress variable. Width of the lines = association strength. Positive interrelations = blue, negative interrelations = red. **Legend:** Frn = friend support, fms = family support, fmc = family cohesion, ngt = negative self-esteem, pst = positive self-esteem, rfl = reflection, brd = brooding, dst = distress tolerance, agg = aggression, exp = expressive suppression, GD = general distress. The boxes depict the maximal interrelation difference between the respective two networks (M), the difference in global network expected influence (EI) between the respective two networks (EI), and the corresponding p-values (5000 comparison samples).

## APPENDIX C.15: SIMILARITY AND DIFFERENCES TO FIGURES IN CHAPTER 3

Several network figures for age 14 (i.e. Figure 2 and 3 in Chapter 4, and figures in Appendix C.2, C.5, C.7, C.9, C.11, C.12, C.14) are similar to figures in our previous report on this sample in Chapter 3 and in J. Fritz, Fried, et al. (2018; Scientific Reports; can be retrieved from https://doi.org/10.1038/s41598-018-34130-2). In the original article, the figures were published under the Creative Commons Attribution 4.0 International License. Information about this license can be found in the article itself (J. Fritz, Fried, et al., 2018) or at http://creativecommons.org/licenses/by/4.0/. The figures here are however only partially similar to the figures in our previous report, for the following reasons: (1) a slightly different sample was used as we could impute the missing data, (2) the general distress variable was not the same for reasons described in Appendix C.1, (3) the brooding variable was not the same for reasons described in Appendix C.1, (4) the scores were not derived from one-factor CFAs, but from longitudinal categorical CFAs with two factors, one for each time point, and (5) due to computing different CFA models, some CFAs did not need the modifications we had to apply for the CFAs in Chapter 3.

## APPENDIX C.16: ARE RESILIENCE AND RISK FACTORS OPPOSING SIDES OF THE SAME COIN?

In the resilience literature there is a sparse but ongoing discourse about whether resilience and risk factors lie on one continuum, representing respectively the opposite ends. Yet, the answer to this question is probably not done justice with a simple yes or no. For our study the resilience factors (RFs) were derived from our systematic review (Chapter 2 or J. Fritz, de Graaff, et al., 2018) and were defined as follows: "*RFs have a promotive impact on the adjustment process following CA and thus help individuals to adapt and recover from the sequelae of CA (Rutter, 1985, 2013; Zolkoski & Bullock, 2012). Statistically, RFs operate as a moderator (Fergus & Zimmerman, 2005; Rutter, 1985), and/ or as a positive mediator (Masten, 2001; van Harmelen et al., 2016) for the relationship between CA and psychopathology.*" We further specified as inclusion criteria that an RF "*belongs either to the individual-, family-, or community-level category, […] belongs to the cognitive, behavioural, social, and/ or emotional functioning domain, [… and should be] amenable"* (see Chapter 2 or J. Fritz, de Graaff, et al., 2018). We specified as exclusion criteria that the RF should not be "*defined (a) as financial advantage, (b) as no re-victimization, (c) as inverse of CA, [and] (d) as inverse of psychopathology*" (see Chapter 2 or J. Fritz, de Graaff, et al., 2018). In other words, RFs are amenable factors that operate as ameliorating or modifying variables in the relationship between adversity and mental health problems, and should neither be equivalent to CA nor to mental health problems. CA was defined as "*traumatic and/or severely stressful events, [leading to]…a higher risk of developing mental health problems*" (see Chapter 2 or J. Fritz, de Graaff, et al., 2018). We additionally specified in detail which events would qualify as CA, in the attempt to keep the definitions of RFs and CA as separate as possible. Yet, based on our definitions, adversity is not equivalent to risk factors, as risk factors do not need to be traumatic and/or severely stressful events but still lead to a higher risk of developing mental health problems (e.g. low maternal education). With regard to our study, the quick, but insufficient answer is probably that many (or most) of the investigated RFs are indeed the flip side of risk factors. For example, self-esteem (or a positive self-concept) is commonly defined as RF and has been discussed as such by many of the seminal resilience researchers, including Michael Rutter, Emmy Werner, Ann Masten, and Michael Ungar (for a review see e.g. Shean, 2015; or Traub & Boynton-Jarrett, 2017). Yet, at the same time a low level of self-esteem or self-worth is part of the DSM V criteria for depression ("Feelings of worthlessness"; American Psychiatric Association, 2013). Hence, whereas a high level of self-esteem may protect against low mood levels, low self-esteem is assumed to contribute to or reflect low mood.

That said, some RFs have been suggested not to lie on the same continuum with their supposingly opposing risk factor. For example, Carretta, Ridner, and Dietrich (2014) showed that hope and hopelessness are highly negatively correlated, but not as high as would be expected for opposing poles of the same construct (Cheavens, Cukrowicz, Hansen, & Mitchell, 2016). Others have proposed that not hopelessness and hope, but hopelessness and the absence of hopelessness may be bipolar (Cheavens et al., 2016; Grewal & Porter, 2007). Hence, here the risk factor may be hopelessness and the RF hope, which may however not lie on the exact same continuum. For other factors the liaison

between risk and protection seems comparably complex, as it depends on the content valence. For example, consider negative and positive rumination. Low negative rumination, called low brooding throughout this thesis, has been solidly found to be linked to a lower level of depression, while high negative rumination has been linked to higher levels of depression (Harding & Mezulis, 2017). Similarly, some research suggests that high positive rumination is linked to a lower level of depression, and particularly to lower levels of anhedonia, while low positive rumination is linked to higher levels of depression or anhedonia (Harding & Mezulis, 2017). Interestingly, negative and positive rumination have been found to be positively related, and have been suggested to overlap in terms of affect amplification, but to neither be the same construct (i.e. the former focusses on negative and the latter on positive content), nor to operate on opposing sides of the same continuum. And for yet other factors the liaison between risk and protection seems even more complicated, as it depends on external aspects. For instance, a low level of expressive suppression, which means that someone can effectively express and communicate his/her emotions, may well be advantageous in safe environments. Yet, in hazardous environments, as for example a violent home environment, emotional expression may not always be advantageous. Similarly, Luthar (1991) found in adolescents from underprivileged environments that high intelligence functions as risk factor, rather than, as commonly found, as RF. Hence some RFs may be protective in one context or environment but may be harmful in another (Shean, 2015). Moreover, some RFs may be particularly protective during early development and others during adulthood. For example, some researchers argue that parental support is particularly protective during childhood, but less so during adolescence (Hostinar, Johnson, & Gunnar, 2015).

Sometimes researchers differentiate between RFs and risk factors through defining risk factors as direct effects, and RFs as moderators or mediators (e.g. see Shaikh & Kauppi, 2010). Such attempts are limited in my opinion, as both mediating and moderating RFs statistically also require a direct effect between the RF and the mental health outcome. Hence, according to such a definition one would suggest that all factors that qualify as direct effect and as mediator and/or moderator should be clustered into the RF category, whereas all factors that only qualify as direct effect should be clustered into the risk factor category. One crucial consideration that limits this definition is the present lack of replicability of RFs and risk factors. For example, Dubow and colleagues (2012) found that positive parenting moderates the relationship between CA and mental health problems, while Cui and Conger (2008) did not find convincing support for a moderation effect. Thus, now one would be stuck with deciding on whether high positive parenting should be considered as RF or whether low positive parenting should be considered as risk factor. Other resilience researchers have argued that both direct effects and mediating and moderating effects qualify as RFs. For example, Garmezy, Masten, and Tellegen (1984) refer to RFs with a direct effect on mental health as “compensatory” factors and to RFs with an interaction effect on mental health as “protective” factors.

In sum, I cautiously conclude that on the group level some RFs may operate on a continuum with risk factors, while for others the relationship with risk seems to be more complex. On an individual level, the relationship between resilience and risk factors is likely to additionally depend on biological predispositions, type of adversity experienced, the specific environmental circumstances, and the developmental stage.

While neither our RF definition nor our analyses allow us to clearly demarcate the conceptualisation of resilience vs risk factors, I believe that our work expands the RF literature on another aspect. Shaik and Kauppi (2010; p. 162-163) state that "*[o]ne of the major shortcomings is the tendency to view factors as mono-directional influences as opposed to bi-directional influences (Glantz & Sloboda, 1999). These models fail to delineate how all factors can be the influences, mediators and outcomes tied in varying degrees to the entire system of variables. Despite the large number of empirical studies […], there are not sufficient details available about how and why the protective or compensatory factors directly or indirectly influence the outcomes (Lepore & Revenson, 2006).*" I believe that the strength of our research lies in shedding light onto the bi-directional system of RFs that are associated with a lower risk of mental distress during early and later adolescence. Regardless of whether resilience and risk factors operate on the same continuum, are inversely correlated but not identical, or are positively correlated but content wise opposing, understanding the nature of RFs seems to have universal appeal as it focuses on what promotes good mental health rather than on what increases mental health problems. Knowledge on the promotion of good mental health in adolescents may not only be of clinical, but also of policy interest, as good mental health in today's youth may result in less mental health problems in tomorrow's adults. Or to put it into Garmezy's words (1987; p. 171): "*Government, by providing protective factors, enables some who would otherwise be lost to a fruitful life to move above the threshold of competence needed to survive in an increasingly complex, technological society.*"

# APPENDIX D

# Appendix Chapter 5

## APPENDIX D.1: VARIABLE PREPARATION: CONFIRMATORY FACTOR ANALYSES

For the confirmatory factor analyses (CFAs) we pooled the factor scores over the 10 result sets (i.e. one for each imputation data set). We used modification indices only when statistically necessary and theoretically defensible. All CFA models fitted reasonably (see Table D.1). For aggression the resulting factor scores were notably poorly distributed and we therefore binarized this variable. The continuous latent distress scores used in Chapter 5 are based on a strongly invariant, categorical CFAs (i.e. L+T+I IM models in Table D.1), to ensure the latent mean comparability between distress at age 14 and age 17. More specifically, we applied the delta parametrization, equated item loadings and item thresholds across the two time points (i.e. age 14 and 17), fixed all item intercepts to 0, the item scales of the first time point to 1, the latent factor mean of the first time point to 0, and the latent factor variance of the first time point to 1.

Table D.1
*(Longitudinal) confirmatory factor analyses conducted with the WLSMV estimator, n = 1188*

| Model | Robust CFI | Robust TLI | Robust RMSEA | RMSEA 90% CI | SRMR | Chisq(df) |
|---|---|---|---|---|---|---|
| ***Friendship support (Goodyer et al., 1989), 5 items, 1 unique item covariance*** | | | | | | |
| **BM** | 0.988 | 0.969 | 0.067 | 0.043– 0.093 | 0.036 | 12.652(4) |
| ***Family support (Epstein et al., 1983), 5 items, 1 unique item covariance*** | | | | | | |
| **BM** | 0.995 | 0.987 | 0.062 | 0.039-0.088 | 0.023 | 9.285(4) |
| ***Family cohesion (Epstein et al., 1983), 7 items, 1 unique item covariance*** | | | | | | |
| **BM** | 0.980 | 0.967 | 0.070 | 0.057-0.085 | 0.042 | 48.773(13) |
| ***Positive self-esteem (Rosenberg, 1965), 5 items, 1 unique item covariance*** | | | | | | |
| **BM** | 0.996 | 0.989 | 0.076 | 0.052-0.102 | 0.016 | 9.446(4) |
| ***Negative self-esteem (Rosenberg, 1965), 5 items, 0 unique item covariances*** | | | | | | |
| **BM** | 0.993 | 0.987 | 0.045 | 0.022-0.069 | 0.017 | 6.542(5) |
| ***Brooding (Treynor et al., 2003), 5 items, 0 unique item covariances*** | | | | | | |
| **BM** | 0.991 | 0.983 | 0.068 | 0.047-0.091 | 0.029 | 14.520(5) |
| ***Reflection (Treynor et al., 2003), 5 items, 1 unique item covariance*** | | | | | | |
| **BM** | 0.999 | 0.998 | 0.023 | 0.000-0.054 | 0.018 | 3.435(4) |
| ***Distress tolerance (Bould et al., 2013), 5 items, 1 unique item covariance*** | | | | | | |
| **BM** | 0.977 | 0.942 | 0.128 | 0.105-0.153 | 0.051 | 36.272(4) |
| ***Aggression (Goodyer et al., 2011), 4 items, 0 unique item covariances*** | | | | | | |
| **BM** | 0.988 | 0.965 | 0.029 | 0.000-0.071 | 0.036 | 1.387(2) |
| ***Distress (Messer et al., 1995; Reynolds & Richmond, 1978), 41 items, 2 unique item covariances*** | | | | | | |
| **C IM 1** | 0.988 | 0.987 | 0.026 | 0.025-0.027 | 0.043 | |
| **C IM 2** | 0.988 | 0.988 | 0.026 | 0.025-0.027 | 0.043 | |
| **C IM 3** | 0.989 | 0.989 | 0.026 | 0.025-0.027 | 0.043 | |
| **C IM 4** | 0.989 | 0.989 | 0.026 | 0.025-0.027 | 0.043 | |
| **C IM 5** | 0.988 | 0.987 | 0.027 | 0.026-0.028 | 0.045 | |
| **C IM 6** | 0.989 | 0.988 | 0.026 | 0.025-0.027 | 0.044 | |
| **C IM 7** | 0.990 | 0.990 | 0.026 | 0.025-0.027 | 0.044 | |
| **C IM 8** | 0.986 | 0.986 | 0.027 | 0.026-0.028 | 0.047 | |
| **C IM 9** | 0.989 | 0.988 | 0.026 | 0.025-0.028 | 0.045 | |
| **C IM 10** | 0.988 | 0.988 | 0.026 | 0.025-0.027 | 0.045 | |
| **L+T+I IM 1** | 0.986 | 0.986 | 0.027 | 0.026-0.028 | 0.042 | |
| **L+T+I IM 2** | 0.987 | 0.987 | 0.027 | 0.026-0.028 | 0.042 | |

| | | | | | |
|---|---|---|---|---|---|
| **L+T+I IM 3** | 0.988 | 0.988 | 0.027 | 0.026-0.028 | 0.042 |
| **L+T+I IM 4** | 0.988 | 0.988 | 0.027 | 0.026-0.028 | 0.043 |
| **L+T+I IM 5** | 0.986 | 0.986 | 0.028 | 0.027-0.029 | 0.045 |
| **L+T+I IM 6** | 0.987 | 0.987 | 0.027 | 0.026-0.028 | 0.043 |
| **L+T+I IM 7** | 0.989 | 0.989 | 0.027 | 0.026-0.028 | 0.043 |
| **L+T+I IM 8** | 0.985 | 0.984 | 0.028 | 0.027-0.029 | 0.046 |
| **L+T+I IM 9** | 0.988 | 0.988 | 0.027 | 0.026-0.028 | 0.044 |
| **L+T+I IM 10** | 0.987 | 0.987 | 0.027 | 0.026-0.029 | 0.044 |

*Note.* WLSMV = weighted least squares mean and variance corrected estimator; CFI = Comparative fit index; TLI = Tucker-Lewis index; RMSEA = Root mean square error of approximation; CI = Confidence interval; chisq = chi-square; BM = baseline model; C IM = configural invariance model; L+T+I IM = loadings, thresholds, and intercepts invariance model. All models were conducted with the delta parameterization.

## APPENDIX D.2: VARIABLE PREPARATION: LATENT CLASS ANALYSIS AND FACTOR MIXTURE MODELS

For the categorical prediction model we aimed to classify the adolescents based on their distress profiles into a categorical distress variable. Firstly, we applied latent class analysis (LCA) with ordinal items, an MLR estimator, and a logit link (see Table D.2), to identify possible class solutions. We used the same 41 anxiety and depression items for the LCA as for the general distress factor model. The 2-class solution was significantly better than a 1-class model (Likelihood-Ratio test (LRT) = 22924.59, $p < .001$). We also tested 3- and 4-class solutions but those did not fit significantly better. Based on those results we conducted a series of factor mixture models (FMMs; Clark et al., 2013), which are hybrid models that add latent classes on top of the latent factors, with different invariance levels between the classes. We tested those FMMs with 2, 3, and 4 classes. The FMM1 is the factor mixture model with the most invariance constraints between classes, as it only allows the factor mean to vary between classes. The FMM1 with 2 classes did not fit better than the FMM1 with 1 class (LRT = 19632.75, $p = .746$; see Table D.3). The FMM1 with 3 classes fitted better than the FMM1 with 2 classes (LRT = 7608.02, $p < .001$). Similarly, the FMM1 with 4 classes fitted better than the FMM1 with 3 classes (LRT = 2157.44, $p < .001$), but had a lower entropy (0.970 vs 0.953). For the analyses with the non-imputed data, one class of the FMM1 4-class model only contained 42 adolescents, with only 10 being sampled in the test sample. This is already a small group to be predicted, but when we then split the sample further into CA+ vs CA- and into female vs male, the high distress class had for the CA- group only 6 adolescents in the training and 2 in the test sample. Similarly, the female group had only 5 adolescents in the training and 1 in the test sample. We therefore considered this class practically too small. We also tested the FMM2 model, in which in addition to the factor mean also the factor variance can vary between classes. The FMM2 solution with 2 classes fitted well. Yet, the FMM2 model could not successfully be fitted on the non-imputed data. We therefore decided not to go forward with the FMM2 models. In sum, we decided to go forward with the FMM1 3-class solution, to keep comparisons with and without imputed data possible and to have sufficiently predictable class sizes. Moreover, the FMM1 with 3 classes revealed a theoretically plausible and practical solution, which is described in the main text. For completeness we also computed the prediction analyses with the FMM1 with 4 classes as outcome variable, which can be found in Appendix D.9.

Importantly, the categorical class solutions are not necessarily ordered categorical, but can be nominal. Consequently, it is not possible to pool over the class solutions of the 10 imputed data sets, as this would not take into account non-ordered class allocations. The FMM1 naturally results in an ordered categorical class solutions, with class-varying factor means. Yet, for the FMM2, for which we allow in addition to the factor mean also the factor variance to vary per class, the solution can be nominal. Similarly, LCA class results can also be content specific, and thus nominal, rather than ordered categorical. Therefore, we computed a grandmedian dataset, for which we took for each score the median value across the 10 imputed datasets. Based on this data set we then performed the LCA and FMM models. Using a grandmedian dataset is disadvantageous, as it does not take into account the

between-imputation variance, yet, it preserves the interpretation of the classes, which was of particular interest here.

Table D.2
*Latent class analyses with MLR estimator and logit link*

| classes | AIC | BIC | $BIC_{adj}$ | Entropy | LMR LRT | p | Class counts |
|---|---|---|---|---|---|---|---|
| 2 | 76466.74 | 77721.51 | 76936.95 | 0.996 | 22924.59 | <.001 | 1=1006; 2=182 |
| 3 | 67746.95 | 69631.64 | 68453.21 | 0.973 | 08957.59 | .765 | 1=647; 2=406; 3=135 |
| 4 | 65288.68 | 67803.29 | 66230.99 | 0.968 | 02703.20 | .767 | 1=159; 2=504; 3=401, 4=124 |

*Note.* AIC =Akaike information criterion. BIC =Bayesian information criterion. $BIC_{adj}$ = sample size adjusted BIC. LMR LRT = Lo-Mendel-Rubin adjusted likelihood ratio test for class comparisons. p = p-value.

Table D.3
*One-factor mixture models with MLR estimator and logit link*

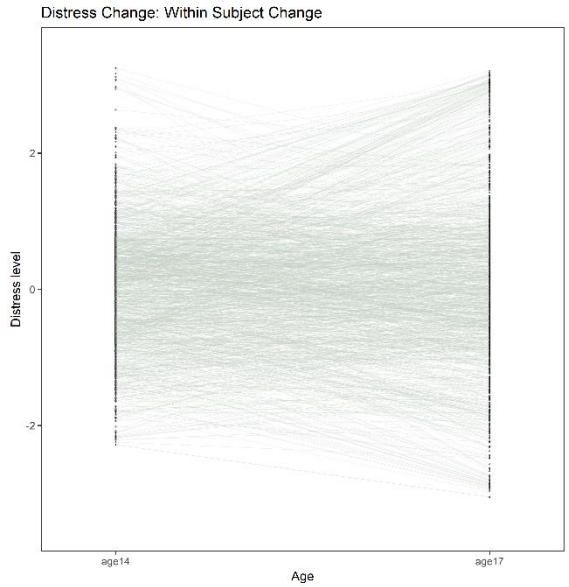
| classes | AIC | BIC | $BIC_{adj}$ | Entropy | LMR LRT | p | Class counts |
|---|---|---|---|---|---|---|---|
| *FMM1: loadings = class invariant; thresholds = class invariant; factor mean = varying per class (fixed to 0 in 1 class for identification); factor variance = fixed to 0* | | | | | | | |
| 2 | 76847.72 | 77685.93 | 77161.82 | 0.998 | 19632.75 | 0.746 | 1=1018; 2=170 |
| 3 | 68169.12 | 69017.49 | 68487.03 | 0.970 | 07608.02 | <0.001 | 1=412; 2=644; 3=132 |
| 4 | 65710.97 | 66569.49 | 66032.68 | 0.953 | 02157.44 | <0.001 | 1=403; 2=480; 3=125; 4=180 |
| *FMM2: loadings = class invariant; thresholds = class invariant; factor mean = varying per class (fixed to 0 in 1 class for identification); factor variance = varying per class* | | | | | | | |
| 2 | 63744.30 | 64592.67 | 64062.21 | 0.989 | 349.03 | <0.001 | 1=1072; 2=116 |
| $3^{NI}$ | - | - | - | 0.977 | - | - | 1=1040; 2=32; 3=116 |
| 4 | 63734.47 | 64613.32 | 64063.80 | 0.585 | 012.47 | 0.578 | 1=657; 2=381; 3=102; 4=48 |

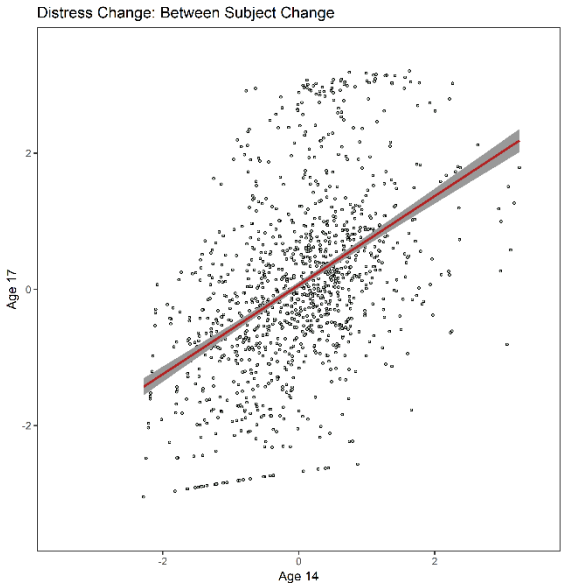
*Note.* AIC =Akaike information criterion. BIC =Bayesian information criterion. $BIC_{adj}$ = sample size adjusted BIC. LMR LRT = Lo-Mendel-Rubin adjusted likelihood ratio test for class comparisons. $^{NI}$ = the model was not identified. p = p-value.

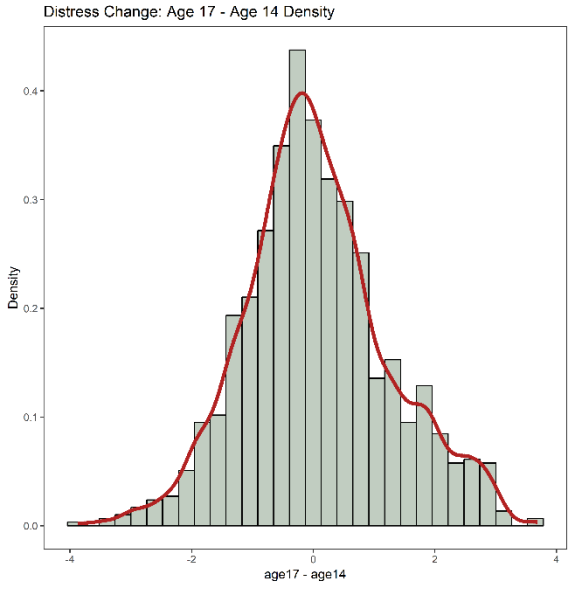
## APPENDIX D.3: OVERVIEW OF USED R PACKAGES, INCLUDING THEIR VERSION NUMBER AND REFERENCE

| Package (version number) | Reference |
|---|---|
| beanplot (1.2) | (Kampstra, 2008) |
| brant (0.2-0) | (Schlegel & Steenbergen, 2018) |
| car (3.0-2) | (Fox & Weisberg, 2011) |
| caret (6.0-81) | (Kuhn, 2018) |
| coin (1.2-2) | (Hothorn et al., 2008) |
| dplyr (0.7.7) | (Wickham et al., 2018) |
| foreign (0.8-70) | (R Core Team, 2017) |
| Hmisc (4.1-1) | (Harrell Jr., 2018) |
| MASS (7.3-50) | (Venables & Ripley, 2002) |
| mice (3.5.0) | (van Buuren & Groothuis-Oudshoorn, 2011) |
| MLmetric (1.1.1) | (Yan, 2016) |
| pastecs (1.3.21) | (Grosjean & Ibanez, 2018) |
| pROC (1.14.0) | (Robin et al., 2011) |
| qgraph (1.5) | (Epskamp et al., 2012) |
| relaimpo (2.2-3) | (Groemping, 2006) |
| reshape (0.8.8) | (Wickham, 2007) |
| semTools (0.5-1.933) | (Jorgensen et al., 2018) |
| VGAM (1.1-1) | (Yee, 2010) |

## APPENDIX D.4: AGE 14 TO AGE 17 CHANGE OF THE MENTAL DISTRESS VARIABLE




The upper left panel shows the within-subject (idiographic) change in distress from age 14 to age 17. Many adolescents have a rather stable trajectory, but others increase or decrease in distress. This largely variable pattern would also be expected in a naturalistic population sample. The upper right panel depicts the between-subject (nomothetic) association between age-14 and age-17 distress. The positive association indicates that on average adolescents with high age-14 distress also have high age-17 distress. The lower left panel shows that change in distress between age 14 and 17 (i.e. age-17 minus age-14 scores) is overall normally distributed. The plots indicate that there is variability in the change of distress that could be explained by resilience factors.

## APPENDIX D.5: TESTING MULTICOLLINEARITY

*Variance inflation factors*

| Mod | CA | gender | Frn | Fms | Fmc | Ngt | Pst | Brd | Rfl | Dst | Agg | Exp | D14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| B2 | 1.00 | 1.00 | | | | | | | | | | | |
| M1 | 1.08 | 1.23 | 1.23 | 1.91 | 2.11 | 2.41 | 1.85 | 2.27 | 1.75 | 1.17 | 1.17 | 1.06 | |
| M2 | 1.02 | 1.06 | | | | | | | | | | | 1.08 |
| M3 | 1.08 | 1.24 | 1.27 | 1.91 | 2.13 | 3.03 | 1.87 | 2.49 | 1.76 | 1.17 | 1.21 | 1.06 | 3.52 |

*Note.* Mod = model; CA = childhood adversity; Frn = friendship support; Fms = family support; Fmc = family cohesion; Ngt = negative self-esteem; Pst = positive self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression; D14 = age-14 distress; B2 = baseline model with CA and gender as predictors; M1 = main model with CA, gender and RFs as predictors; M2 = main model with CA, gender and age-14 distress as predictors; M3 = main model with CA, gender, RFs and age-14 distress as predictors. When taking the square root of the variance inflation factors, none is bigger than 2, which additionally underpins the absence of multicollinearity.

## APPENDIX D.6: TESTING AND RELAXING THE PARALLEL SLOPES ASSUMPTION

Ordered categorical, or proportional odds models, have a “proportional odds” or also called “parallel slopes” assumption. This assumption necessitates that when the tested ordinal categories are dichotomized (e.g. here “a”: low vs moderate and high, and “b”: low and moderate vs high) the logistic prediction of the respective dichotomized categories results in two slopes (i.e. one for scenario “a” and one for scenario “b”) that do not differ significantly from each other. If the slopes differ significantly, the proportional odds assumption does not hold and needs to be relaxed. The assumption can be determined for each predictor in the model and only for those predictors that do not meet the assumption separate slope values need to be estimated. This then results in a partial proportional odds model. It would also be possible to estimate a non-proportional odds model to circumvent the assumption for every variable in the model. However, this would be highly disadvantageous as it requires a vast amount of power. Hence we opted for the partial proportional odds model to ensure that we have as much power as possible. Table D.4 depicts all the variables for which the proportional odds assumption was relaxed:

Table D.4
*Variables for which the proportional odds assumption was relaxed*

| | **M1: RFs only** | **M2: D14 only** | **M3: RFs & D14** |
|---|---|---|---|
| **Whole sample** | | | |
| 3-class models | -gender<br>-distress tolerance | -gender<br>-D14 | -gender<br>-distress tolerance |
| 3-class (models with reduced number of RFs) | -gender<br>-brooding | - | -gender |
| 4-class model | -gender | -gender | -gender |
| **CA+ sample** | | | |
| 3-class models | -gender | -gender<br>-D14 | -gender<br>-D14 |
| 3-class (models with reduced number of RFs) | -gender<br>-brooding | - | -gender<br>-D14 |
| 4-class model | -gender | -gender<br>-D14 | -gender<br>-D14 |
| **CA- sample** | | | |
| 3-class models | -gender | -gender | -gender<br>-distress tolerance |
| 3-class (models with reduced number of RFs) | -gender | - | -gender |
| 4-class model | -gender<br>-reflection | -gender | -gender<br>-reflection |
| **Female sample** | | | |
| 3-class models | -distress tolerance | -D14 | -distress tolerance |
| 3-class (models with reduced number of RFs) | -distress tolerance | - | -distress tolerance |
| 4-class model | x | -D14 | -D14 |
| **Male sample** | | | |
| 3-class models | -friendship support<br>-aggression | -D14 | -aggression<br>-D14 |

| | | | |
|---|---|---|---|
| 3-class (models with reduced number of RFs) | -negative self-esteem<br>-aggression | - | -aggression<br>-D14 |
| 4-class model | -reflection<br>-brooding | -D14 | -reflection<br>-D14 |

*Note.* RFs = resilience factors, D14 = distress at age 14. – means not tested. X means that all variables met the proportional odds assumption.

## APPENDIX D.7: COMPARING MODEL ACCURACIES BETWEEN CHILDHOOD ADVERSITY AND GENDER SUBGROUPS

The below tables depict the prediction accuracy of the prediction models described in Chapter 5. Table D.5 and D.6 depict subgroup accuracy comparisons for CA and gender models, respectively. Table D.7 depicts accuracy comparisons for models including all RFs versus models that only include a subset of the RFs.

Table D.5
*Subgroup accuracy comparisons for childhood adversity (CA) models*

| Model | Coefficient | CA+ | CA- | Proportion test summary |
|---|---|---|---|---|
| M1 ordinal | Accuracy | 54% | 66% | Chi-squared = 3.8242, df = 1, |
| | Correct predictions | 84 | 82 | p-value = 0.051 |
| | Total predictions | 156 | 124 | |
| M2 ordinal | Accuracy | 60% | 69% | Chi-squared = 2.1104, df = 1, |
| | Correct predictions | 94 | 86 | p-value = 0.146 |
| | Total predictions | 156 | 124 | |
| M3 ordinal | Accuracy | 58% | 69% | Chi-squared = 2.6657, df = 1, |
| | Correct predictions | 91 | 85 | p-value = 0.103 |
| | Total predictions | 156 | 124 | |
| M1 linear | Accuracy | 34.62% | 40.32% | Chi-squared = 0.73489, df = 1, |
| | Correct predictions | 54 | 50 | p-value = 0.391 |
| | Total predictions | 156 | 124 | |
| M2 linear | Accuracy | 32.69% | 38.71% | Chi-squared = 0.84703, df = 1, |
| | Correct predictions | 51 | 48 | p-value = 0.357 |
| | Total predictions | 156 | 124 | |
| M3 linear | Accuracy | 36.54% | 38.71% | Chi-squared = 0.06176, df = 1, |
| | Correct predictions | 57 | 48 | p-value = 0.804 |
| | Total predictions | 156 | 124 | |
| M1 ordinal | Accuracy | 47% | 51% | Chi-squared = 0.29897, df = 1, |
| 4 classes | Correct predictions | 73 | 63 | p-value = 0.585 |
| | Total predictions | 156 | 124 | |
| M2 ordinal | Accuracy | 56% | 57% | Chi-squared = 7.0965e-31, df = 1, |
| 4 classes | Correct predictions | 87 | 70 | p-value = 1 |
| | Total predictions | 156 | 124 | |
| M3 ordinal | Accuracy | 48% | 54% | Chi-squared = 0.75649, df = 1, |
| 4 classes | Correct predictions | 75 | 67 | p-value = 0.384 |
| | Total predictions | 156 | 124 | |

*Note.* CA+ = adolescents with CA, CA- = adolescents without CA, RFs = resilience factors, M1 = Model 1 contains the ten RFs, M2 = Model 2 contains age-14 distress, M3 = Model 3 contains both the RFs and age-14 distress as predictors for age-17 distress. Correct predictions = number of correctly predicted adolescents, Total predictions = number of adolescents that could have been predicted correctly, Accuracy = ratio correct predictions divided by total predictions. df = degrees of freedom.

Table D.6
*Subgroup accuracy comparisons for gender models*

| Model | Coefficient | female | male | Proportion test summary |
|---|---|---|---|---|
| M1 ordinal | Accuracy | 58% | 61% | Chi-squared = 0.29082, df = 1, |
| | Correct predictions | 88 | 78 | p-value = 0.590 |
| | Total predictions | 153 | 127 | |
| M2 ordinal | Accuracy | 59% | 64% | Chi-squared = 0.52365, df = 1, |
| | Correct predictions | 90 | 81 | p-value = 0.469 |
| | Total predictions | 153 | 127 | |

| | | | | |
|---|---|---|---|---|
| M3 ordinal | Accuracy | 58% | 61% | Chi-squared = 0.18409, df = 1, |
| | Correct predictions | 89 | 78 | p-value = 0.668 |
| | Total predictions | 153 | 127 | |
| M1 linear | Accuracy | 32.90% | 38.89% | Chi-squared = 0.83392, df = 1, |
| | Correct predictions | 50 | 49 | p-value = 0.361 |
| | Total predictions | 152 | 126 | |
| M2 linear | Accuracy | 35.53% | 41.27% | Chi-squared = 0.73526, df = 1, |
| | Correct predictions | 54 | 52 | p-value = 0.391 |
| | Total predictions | 152 | 126 | |
| M3 linear | Accuracy | 34.87% | 42.06% | Chi-squared = 1.2222, df = 1, |
| | Correct predictions | 53 | 53 | p-value = 0.269 |
| | Total predictions | 152 | 126 | |
| M1 ordinal | Accuracy | 43% | 48% | Chi-squared = 0.4571, df = 1, |
| 4 classes | Correct predictions | 66 | 60 | p-value = 0.499 |
| | Total predictions | 154 | 126 | |
| M2 ordinal | Accuracy | 53% | 53% | Chi-squared = 1.6826e-30, df = 1, |
| 4 classes | Correct predictions | 81 | 67 | p-value = 1 |
| | Total predictions | 154 | 126 | |
| M3 ordinal | Accuracy | 49% | 52% | Chi-squared = 0.14779, df = 1, |
| 4 classes | Correct predictions | 76 | 66 | p-value = 0.701 |
| | Total predictions | 154 | 126 | |

*Note.* RFs = resilience factors, M1 = Model 1 contains the ten RFs, M2 = Model 2 contains age-14 distress, M3 = Model 3 contains both the RFs and age-14 distress as predictors for age-17 distress. Correct predictions = number of correctly predicted adolescents, Total predictions = number of adolescents that could have been predicted correctly, Accuracy = ratio correct predictions divided by total predictions. df = degrees of freedom.

Table D.7

*Accuracy comparison for models including all resilience factors (RFs) versus those including a subset of the RFs*

| **Model** | **Coefficient** | **All RFs** | **3 RFs** | **Proportion test summary** |
|---|---|---|---|---|
| M1 ordinal | Accuracy | 64% | 62% | Chi-squared = 0.374, df = 1, |
| | Correct predictions | 181 | 173 | p-value = 0.541 |
| | Total predictions | 281 | 281 | |
| M3 ordinal | Accuracy | 63% | 60% | Chi-squared = 0.6091, df = 1, |
| | Correct predictions | 178 | 168 | p-value = 0.435 |
| | Total predictions | 281 | 281 | |
| M1 linear | Accuracy | 37.14% | 37.14% | Chi-squared = 0, df = 1, |
| | Correct predictions | 104 | 104 | p-value = 1 |
| | Total predictions | 280 | 280 | |
| M3 linear | Accuracy | 40.71% | 40.71% | Chi-squared = 0, df = 1, |
| | Correct predictions | 114 | 114 | p-value = 1 |
| | Total predictions | 280 | 280 | |

*Note.* M1 = Model 1 contains the RFs, M3 = Model 3 contains both the RFs and age-14 distress as predictors for age-17 distress. Correct predictions = number of correctly predicted adolescents, Total predictions = number of adolescents that could have been predicted correctly, Accuracy = ratio correct predictions divided by total predictions. df = degrees of freedom.

## APPENDIX D.8: PREDICTION ANALYSES WITH SUBSETS OF THE RESILIENCE FACTORS, IN THE CHILDHOOD ADVERSITY AND GENDER SUBGROUPS

For the CA+ group we tested six RFs in addition to gender, as those were significant in the multivariable model, namely: friendship support, family cohesion, positive self-esteem, brooding, distress tolerance, and aggression. Those models were similarly predictive as the models with all 10 RFs and gender (change in accuracy: ordinal models from 54% to 58%; linear models from 34.62% to 33.97%). We also tested those two models while additionally including age-14 distress, which were again similar as the models with gender, the 10 RFs and age-14 distress (change in accuracy: ordinal models from 58% to 60%; linear models from 36.54% to 35.90%).

For the CA- group we tested four RFs in addition to gender, as those were significant in the multivariable model, namely: family support, positive self-esteem, negative self-esteem, and brooding. Those models were similarly predictive as the models with all 10 RFs and gender (change in accuracy: ordinal models from 66% to 65%; linear models from 40.32% to 41.94%). We also tested those two models while additionally including age-14 distress, which were again similar as the models with gender, the 10 RFs and age-14 distress (change in accuracy: ordinal models from 69% to 68%; linear models from 38.71% to 37.10%).

For female adolescents we tested three RFs, as those were significant in the multivariable model, namely: negative self-esteem, brooding and distress tolerance. Those models were similarly predictive as the models with all 10 RFs and CA (change in accuracy: ordinal models from 58% to 56%; linear models from 32.90% to 31.58%). We also tested those two models while additionally including age-14 distress, which were again similar as the models with CA, the 10 RFs and age-14 distress (change in accuracy: ordinal models from 58% to 58%; linear models from 34.87% to 36.84%).

For male adolescents we tested three RFs, as those were significant in the multivariable model, namely: positive self-esteem, negative self-esteem and aggression. Those models were similarly predictive as the models with all 10 RFs and CA (change in accuracy: ordinal models from 61% to 61%; linear models from 38.89% to 43.65%). We also tested those two models while additionally including age-14 distress, which were again similar as the models with CA, 10 RFs and age-14 distress (change in accuracy: ordinal models from 61% to 61%; linear models from 42.06% to 38.89%).

Here we did not test whether the accuracy differed significantly between the subgroups (i.e. CA+ vs CA-, and female vs male) as we tested the subgroups with different sets of RF predictors.

## APPENDIX D.9: ORDINAL PREDICTION ANALYSES WITH A 4-CLASS DISTRESS OUTCOME VARIABLE

Similar to the 3-class model, the 4-class model revealed a plausible distress severity solution, split in a low, low/moderate, moderate/high and a high distress severity class (see Figure D.1). We also conducted three *ordinal* prediction models with the 4-class distress variable as ordered categorical outcome variable. Of the three models one again contained the RFs (M1), one age-14 distress (M2) and one both (RFs and age-14 distress; M3) in addition to gender and CA. The three models had a low accuracy ranging from 50% to 52% (see Table D.8), resulting for all three models in about 1 in 2 adolescents who were predicted into their correct distress severity class. The results were comparable when we split the adolescents into CA+ (accuracy: M1 = 47%, M2 = 56%, M3 = 48%), CA- (accuracy: M1 = 51%, M2 = 57%, M3 = 54%), female (accuracy: M1 = 43%, M2 = 53%, M3 = 49%) and male groups (accuracy: M1 = 48%, M2 = 53%, M3 = 52%). Moreover, the prediction accuracy did not differ significantly between the CA and gender subgroups (see Appendix D.7).

Table D.8
*Ordinal prediction analyses for the whole group: for resilience factors (RFs) only (M1), age-14 distress (D14) only (M2), and RFs and D14 together (M3)*

| | **M1: RFs only** | | **M2: D14 only** | | **M3: RFs & D14** | |
|---|---|---|---|---|---|---|
| | **observed** | **predicted** | **observed** | **predicted** | **observed** | **predicted** |
| Residual deviance | 1903.68 | - | 1849.99 | - | 1836.02 | - |
| ROC | - | low=0.70<br>l/m=0.62<br>m/h=0.69<br>high=0.68 | - | low=0.71<br>l/m=0.63<br>m/h=0.70<br>high=0.71 | - | low=0.71<br>l/m=0.61<br>m/h=0.69<br>high=0.70 |
| Sensitivity | - | low=0.79<br>l/m=0.46<br>m/h=0.07<br>high=0.04 | - | low=0.74<br>l/m=0.58<br>m/h=0.10<br>high=0.04 | - | low=0.71<br>l/m=0.56<br>m/h=0.12<br>high=0.07 |
| Specificity | - | low=0.61<br>l/m=0.65<br>m/h=0.97<br>high=0.98 | - | low=0.69<br>l/m=0.62<br>m/h=0.98<br>high=0.97 | - | low=0.67<br>l/m=0.62<br>m/h=0.98<br>high=0.97 |
| Accuracy | - | 0.50<br>low=0.70<br>l/m=0.56<br>m/h=0.52<br>high=0.51 | - | 0.52<br>low=0.71<br>l/m=0.60<br>m/h=0.54<br>high=0.51 | - | 0.51<br>low=0.69<br>l/m=0.59<br>m/h=0.55<br>high=0.52 |
| Low distress severity | 116 | 155 of which<br>- 91 correct<br>- 46 false l/m<br>- 09 false m/h<br>- 09 false high | 116 | 138 of which<br>- 86 correct<br>- 35 false l/m<br>- 05 false m/h<br>- 12 false high | 116 | 137 of which<br>- 82 correct<br>- 37 false l/m<br>- 07 false m/h<br>- 11 false high |
| Low/mod severity | 96 | 108 of which<br>- 44 correct<br>- 24 false low<br>- 26 false h/m<br>- 14 false high | 96 | 126 of which<br>- 56 correct<br>- 27 false low<br>- 30 false h/m<br>- 13 false high | 96 | 124 of which<br>- 54 correct<br>- 32 false low<br>- 25 false h/m<br>- 13 false high |
| Mod/high severity | 42 | 11 of which<br>- 03 correct<br>- 00 false low<br>- 05 false l/m<br>- 03 false high | 42 | 9 of which<br>- 04 correct<br>- 01 false low<br>- 03 false l/m<br>- 01 false high | 42 | 10 of which<br>- 05 correct<br>- 01 false low<br>- 03 false l/m<br>- 01 false high |
| High distress severity | 27 | 7 of which<br>- 01 correct | 27 | 8 of which<br>- 01 correct | 27 | 10 of which<br>- 02 correct |

| | | |
|---|---|---|
| - 01 false low | - 02 false low | - 01 false low |
| - 01 false l/m | - 02 false l/m | - 02 false l/m |
| - 04 false m/h | - 03 false m/h | - 05 false m/h |

*Note.* Mod = moderate. All models were computed with childhood adversity and gender as predictors. ROC = receiver operating characteristic. Accuracy = relative number of correctly predicted cases. Sensitivity = e.g. for low distress: the number of adolescents who are correctly predicted into the low distress group divided by all adolescent who are actually in the low distress group. Specificity = e.g. for low distress: the number of adolescents who are correctly not predicted into the low distress group divided by all adolescent who are actually not in the low distress group. Variable for which the proportional odds assumption was relaxed can be found in Appendix D.6.

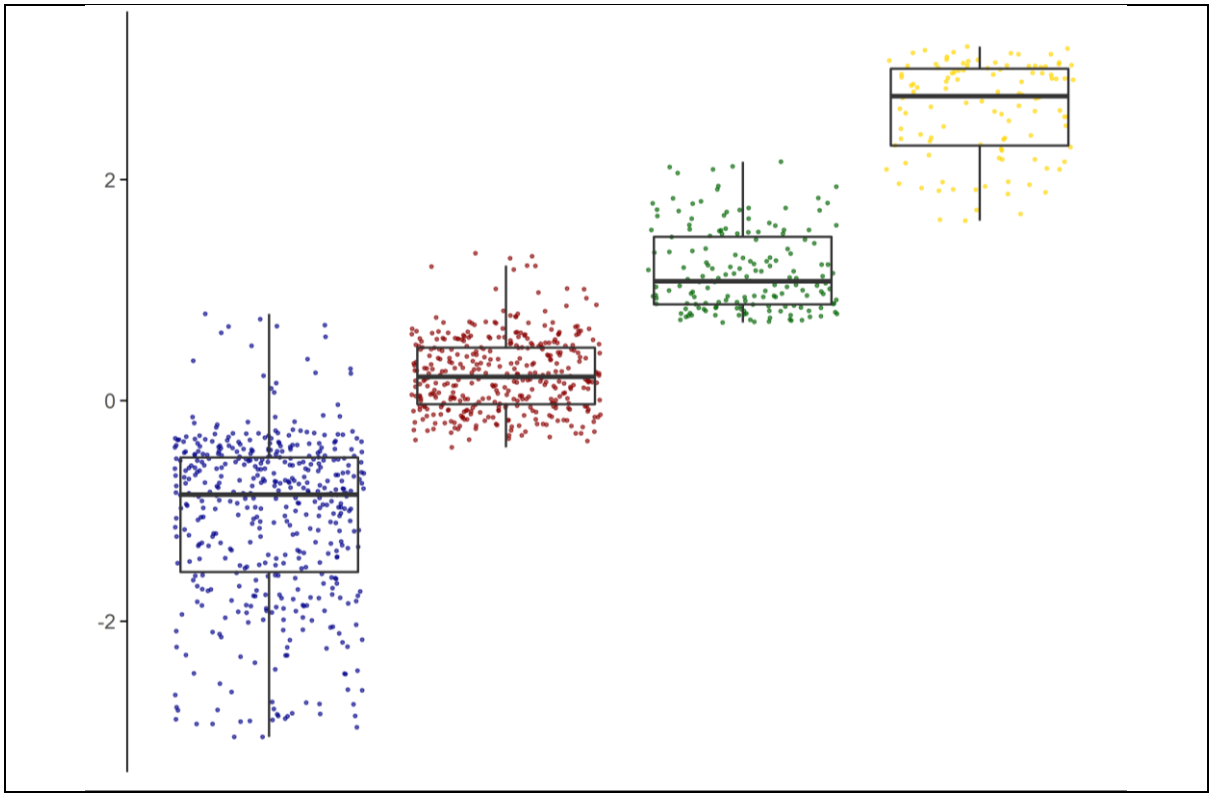



*Figure D.1.* Four-class distress solution (low: n = 466; low/moderate: n = 386; moderate/high: n = 168; high = 110) plotted against the continuous distress severity scores. Center line = median (50% quantile); lower box limit =25% quantile; upper box limit = 75% quantile; lower whisker = smallest observation greater than or equal to the lower box limit -1.5 x Inter Quartile Range (IQR); upper whisker = largest observation less than or equal to the upper box limit + 1.5 x Inter Quartile Range (IQR).

# APPENDIX E

# Appendix Chapter 6

## APPENDIX E.1: RESULTS WITHOUT GENDER CORRECTION

This appendix contains the same set of results as Chapter 6, this time however without gender correction. The below Table E.1 (single main effects) corresponds to Table 2 in the Chapter. The below Table E.2 (single moderation and mediation effects) corresponds to Table 3 in the Chapter. The moderation results of Table E.2 are also depicted in Figure E.1 and the mediation results in Figure E.2. The below Table E.3 (multiple main effects) corresponds to Table 4 in the Chapter. Results for multiple moderation and mediation effects can be found in Appendix E.5.

The mediation Figure 6.1.b2, as well as below Figure E.2, are modelled using an adapted script from M. S. Fritz and MacKinnon (2008a; Behavior Research Methods: https://doi.org/10.3758/BRM.40.1.55), the original scripts can be found in the belonging supplement material (M. S. Fritz & MacKinnon, 2008b, at: https://link.springer.com/article/10.3758/BRM.40.1.55#SupplementaryMaterial).

Table E.1
*Single main effects of the RFs without correction for gender, for the whole sample, the CA+ and the CA- group; corrected for multiple testing*

| | ***Whole sample*** | | | | | ***CA+*** | | | | ***CA-*** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | b | b* | SE | **p** | $R^2$ | b | SE | **p** | $R^2$ | b | SE | **p** | $R^2$ |
| CA | 0.44 | - | .08 | **<0.001*** | 03 | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** |
| Frn | -0.35 | -0.33 | .05 | **<0.001*** | 05 | -0.36 | .06 | **<0.001*** | 05 | -0.29 | .07 | **<0.001*** | 03 |
| Fms | -0.31 | -0.28 | .04 | **<0.001*** | 04 | -0.28 | .06 | **<0.001*** | 04 | -0.30 | .06 | **<0.001*** | 04 |
| Fmc | -0.39 | -0.35 | .04 | **<0.001*** | 07 | -0.38 | .06 | **<0.001*** | 06 | -0.31 | .07 | **<0.001*** | 04 |
| Pst | -0.47 | -0.44 | .04 | **<0.001*** | 11 | -0.43 | .05 | **<0.001*** | 09 | -0.47 | .06 | **<0.001*** | 11 |
| Ngt | -0.60 | -0.57 | .05 | **<0.001*** | 14 | -0.50 | .06 | **<0.001*** | 10 | -0.69 | .07 | **<0.001*** | 18 |
| Brd | -0.50 | -0.48 | .04 | **<0.001*** | 11 | -0.45 | .06 | **<0.001*** | 09 | -0.52 | .06 | **<0.001*** | 12 |
| Rfl | -0.32 | -0.31 | .05 | **<0.001*** | 04 | -0.27 | .06 | **<0.001*** | 03 | -0.36 | .07 | **<0.001*** | 06 |
| Dst | -0.26 | -0.23 | .04 | **<0.001*** | 03 | -0.30 | .06 | **<0.001*** | 04 | -0.14 | .06 | **<0.05*** | 01 |
| Agg | -0.25 | -0.23 | .04 | **<0.001*** | 04 | -0.22 | .05 | **<0.001*** | 03 | -0.25 | .06 | **<0.001*** | 03 |
| Exp | 0.01 | 0.03 | .04 | 0.72 | 00 | 0.05 | .05 | 0.36 | 00 | 0.01 | .06 | 0.80 | 00 |
| D14 | 0.65 | 0.63 | .04 | **<0.001*** | 22 | 0.61 | .05 | **<0.001*** | 19 | 0.67 | .05 | **<0.001*** | 24 |

*Note.* b = unstandardized regression (slope) coefficient, b* = unstandardized regression (slope) coefficient corrected for CA, SE = standard error, p = p-value, $R^2$ = variance explained, represented here as percentage. Of note, SE, p and $R^2$ belong to the effects that are not corrected for CA. CA = childhood adversity, CA+ = adolescents with CA, CA- = adolescents without CA; Frn = friendship support; Fms = family support; Fmc = family cohesion; Pst = positive self-esteem; Ngt = negative self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression; D14 = age-14 distress.

Table E.2
*Single moderation and mediation effects of the RFs without correction for gender; corrected for multiple testing*

| | Moderation: interaction effect | | | | Mediation: indirect effect | | | |
|---|---|---|---|---|---|---|---|---|
| | b | SE | p | %$R^2$ | b | SE | p | MC CI |
| Frn | -0.06 | 0.10 | 0.79 | 07 | 0.04 | 0.02 | **<0.01*** | .01-.08 |
| Fms | 0.02 | 0.09 | 0.83 | 07 | 0.05 | 0.02 | **<0.01*** | .02-.08 |
| Fmc | -0.07 | 0.09 | 0.78 | 08 | 0.13 | 0.02 | **<0.001*** | .08-.18 |
| Pst | 0.05 | 0.08 | 0.79 | 13 | 0.13 | 0.03 | **<0.001*** | .08-.18 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Ngt | 0.20 | 0.09 | 0.30 | 15 | 0.11 | 0.03 | **<0.001*** | .06-.17 |
| Brd | 0.07 | 0.09 | 0.78 | 13 | 0.07 | 0.03 | **<0.01*** | .03-.12 |
| Rfl | 0.09 | 0.09 | 0.78 | 07 | 0.02 | 0.02 | 0.23 | -.01-.05 |
| Dst | -0.17 | 0.09 | 0.30 | 06 | 0.06 | 0.02 | **<0.001*** | .03-.09 |
| Agg | 0.03 | 0.08 | 0.82 | 06 | 0.05 | 0.02 | **<0.01*** | .02-.09 |
| Exp | 0.03 | 0.08 | 0.82 | 03 | -0.01 | 0.01 | 0.41 | -.02-.01 |
| D14 | -0.06 | 0.07 | 0.78 | 23 | 0.17 | 0.04 | **<0.001*** | .10-.25 |

*Note.* b = unstandardized regression (slope) coefficient, SE = standard error, p = p-value, %$R^2$ = percentage variance explained. MC CI = Monetcarlo confidence interval. Frn = friendship support; Fms = family support; Fmc = family cohesion; Pst = positive self-esteem; Ngt = negative self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression; D14 = age-14 distress.

Table E.3

*Multiple main effects of the RFs without correction for gender, for the whole sample, CA+ and the CA- group*

| | ***Whole sample*** | | | | ***CA+*** | | | ***CA-*** | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | b | b* | SE | p | b | SE | p | b | SE | p |
| CA | 0.25 | - | .07 | **<0.001*** | - | - | - | - | - | - |
| Frn | -0.07 | -0.07 | .05 | 0.15 | -0.15 | .07 | **<0.05*** | 0.04 | .08 | 0.56 |
| Fms | -0.03 | -0.05 | .06 | 0.55 | 0.02 | .08 | 0.85 | -0.14 | .08 | **0.08** |
| Fmc | -0.12 | -0.09 | .06 | **<0.05*** | -0.17 | .08 | **<0.05*** | 0.02 | .08 | 0.78 |
| Pst | -0.18 | -0.17 | .05 | **<0.001*** | -0.19 | .07 | **<0.01*** | -0.15 | .07 | **<0.05*** |
| Ngt | -0.20 | -0.21 | .07 | **<0.01*** | -0.06 | .09 | 0.50 | -0.41 | .10 | **<0.001*** |
| Brd | -0.18 | -0.18 | .06 | **<0.01*** | -0.17 | .09 | **<0.05*** | -0.18 | .09 | **<0.05*** |
| Rfl | -0.01 | -0.02 | .06 | 0.81 | 0.01 | .08 | 0.91 | -0.09 | .08 | 0.26 |
| Dst | -0.08 | -0.06 | .04 | **0.07** | -0.13 | .06 | **<0.05*** | 0.01 | .06 | 0.82 |
| Agg | -0.11 | -0.10 | .04 | **<0.01*** | -0.13 | .05 | **<0.01*** | -0.06 | .06 | 0.35 |
| Exp | 0.05 | 0.06 | .04 | 0.14 | 0.06 | .05 | 0.26 | 0.05 | .05 | 0.31 |
| % $R^2$ | | | | 19% | | | 17% | | | 21% |

*Note.* b = unstandardized regression (slope) coefficient, b* = unstandardized regression (slope) coefficient corrected for CA, SE = standard error, p = p-value. Of note, SE, p and $R^2$ belong to the effects that are not corrected for CA. CA = childhood adversity, CA+ = adolescents with CA, CA- = adolescents without CA; Frn = friendship support; Fms = family support; Fmc = family cohesion; Pst = positive self-esteem; Ngt = negative self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression.

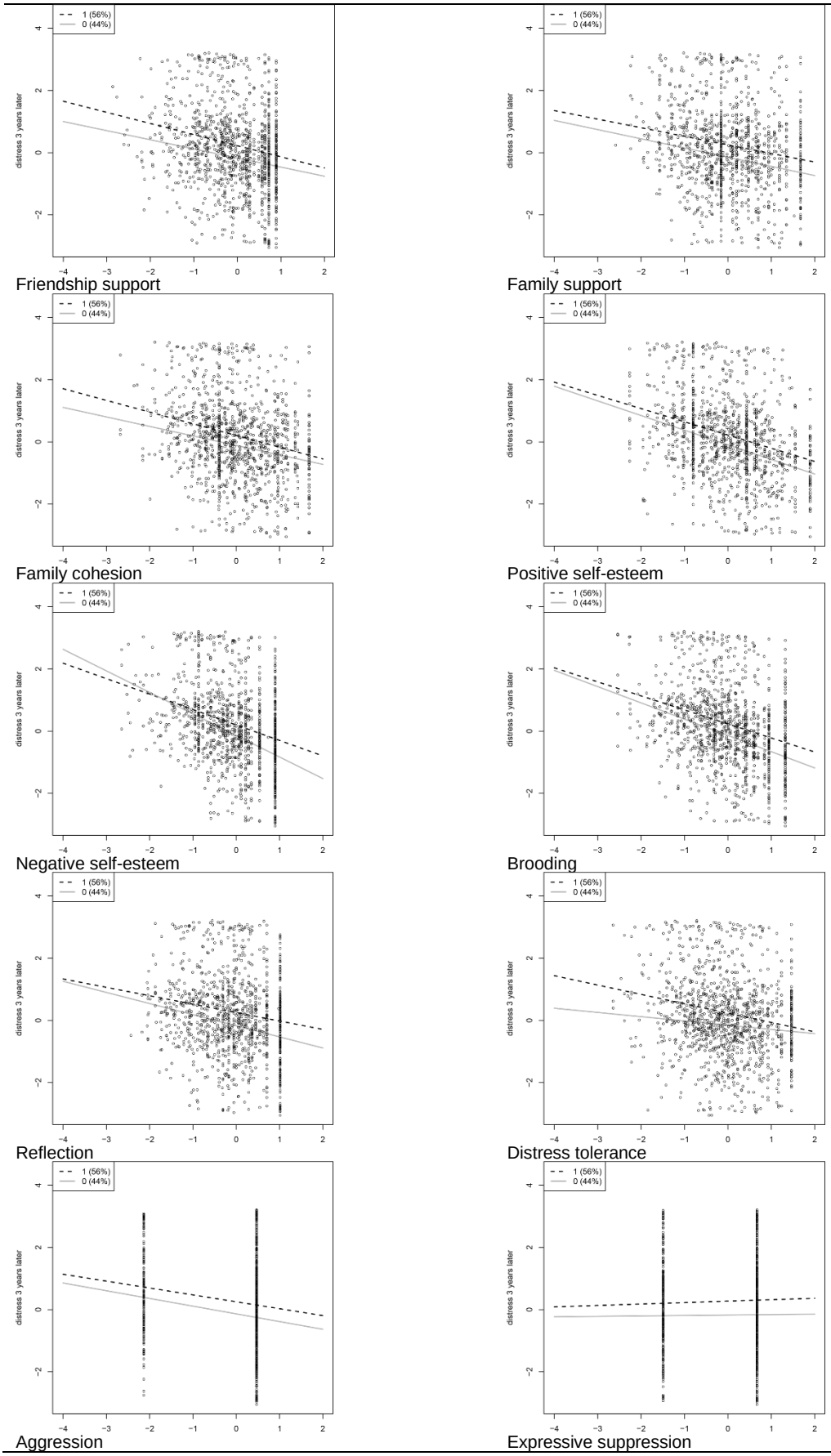



*Figure E.1.* The moderation effects of the RFs for the relationship between CA and age-17-distress. Dashed line = CA+, solid line = CA-.

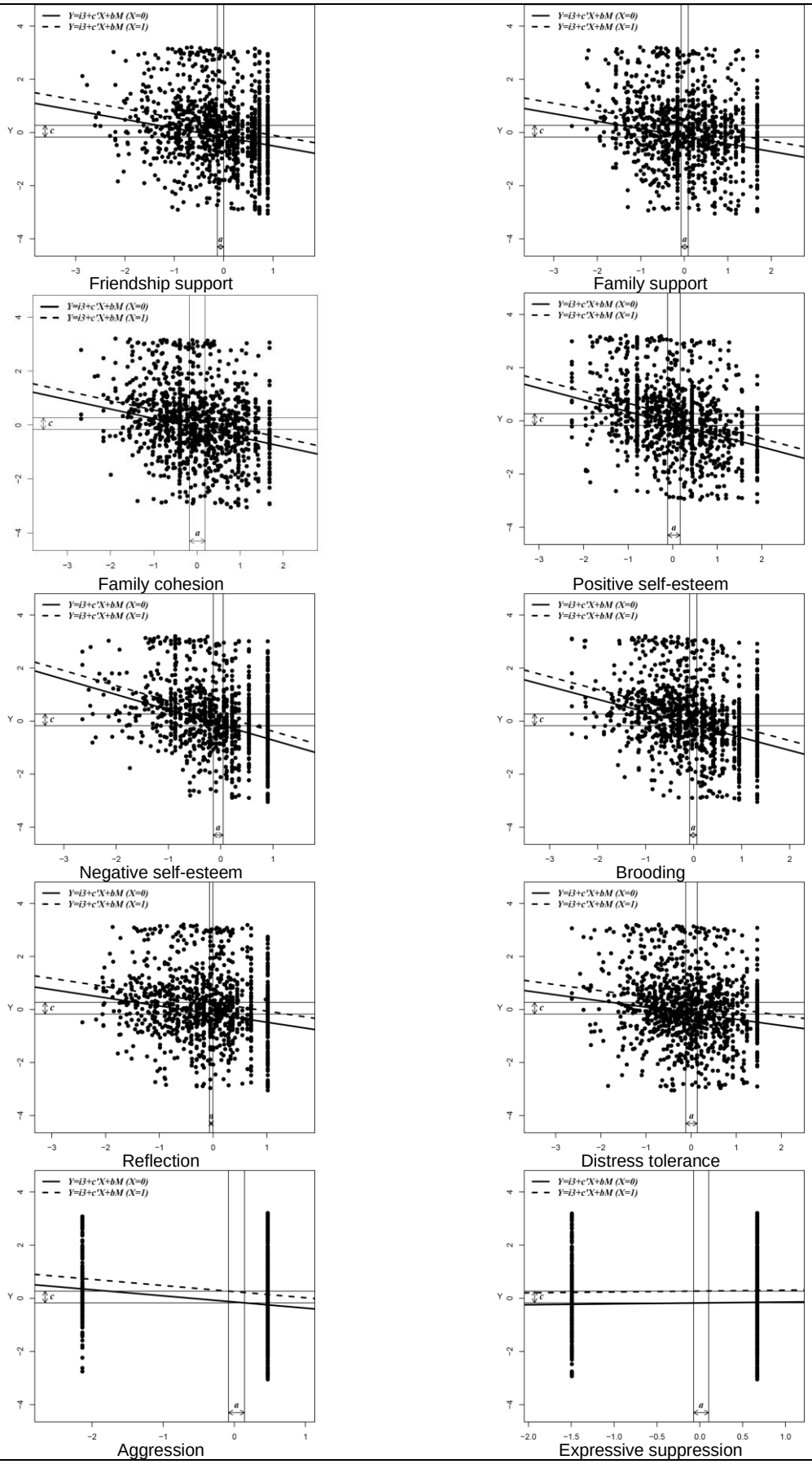



*Figure E.2.* The mediation effects of the RFs for the relationship between CA and age-17-distress. Dashed line = CA+, solid line = CA-.

## APPENDIX E.2: OVERVIEW OF USED R PACKAGES, INCLUDING THEIR VERSION NUMBER AND REFERENCE

| Package (version number) | Reference |
|---|---|
| car (3.0-2) | (Fox & Weisberg, 2011) |
| dplyr (0.7.7) | (Wickham et al., 2018) |
| foreign (0.8-70) | (R Core Team, 2017) |
| lavaan (0.6-4) | (Rosseel, 2012) |
| mice (3.5.0) | (van Buuren & Groothuis-Oudshoorn, 2011) |
| pastecs (1.3.21) | (Grosjean & Ibanez, 2018) |
| rockchalk (1.8.129) | (Johnson, 2018) |
| semTools (0.5-1.933) | (Jorgensen et al., 2018) |
| sjPlot (2.6.2) | (Lüdecke, 2018) |

## APPENDIX E.3: EXPECTED AND ACHIEVED POWER

In line with our preregistered review, we would have expected a low interaction effect size for the moderation effect of the RF and CA (i.e. $f^2 = 0.01$). Given the total sample size of 1130, and α/β ratio of 1, three predictors in total (2 main effects, 1 interaction effect) and 1 predictor being tested (i.e. the interaction effect) our analyses would have had a high amount of power (= .94). For the indirect effect of the mediation models, we would have assumed a moderate, positive correlation between CA and distress at age 17 (~ +0.30), a small, negative correlation between CA and the RF (~ -0.10), and a moderate, negative correlation between the RF and distress at age 17 (~ -0.30). We specified a standard deviation of 1 for the RF and age-17 distress, as most of our RFs and the distress scores were derived form a standardized distribution, and a standard deviation of 0.5 for the CA variable as this one is binary (alongside, 1000 replications with 100000 Monte Carlo draws, a seed of 4444 and 95% confidence interval). Accordingly, the indirect mediation effects would also have had a high amount of power (= .91). We additionally computed the post-hoc power that actually was achieved, which can be found in Table E.4 and clearly reflects our results pattern.

Table E.4
*Expected and achieved power*

| | Moderation | Mediation |
|---|---|---|
| Friendship support | $\triangle R^2 = 0.0004$; 0.10 | 0.80*** |
| Family support | $\triangle R^2 = 0.0000$; 0.06 | 0.88*** |
| Family cohesion | $\triangle R^2 = 0.0005$; 0.12 | 1.00*** |
| Positive self-esteem | $\triangle R^2 = 0.0003$; 0.09 | 1.00*** |
| Negative self-esteem | $\triangle R^2 = 0.0034$; 0.50* | 0.98*** |
| Brooding | $\triangle R^2 = 0.0005$; 0.12 | 0.84*** |
| Reflection | $\triangle R^2 = 0.0007$; 0.15 | 0.26 |
| Distress tolerance | $\triangle R^2 = 0.0031$; 0.46 | 1.00*** |
| Aggression | $\triangle R^2 = 0.0001$; 0.06 | 0.97*** |
| Expressive suppression | $\triangle R^2 = 0.0002$; 0.07 | 0.12 |
| Age-14 distress | $\triangle R^2 = 0.0005$; 0.12 | 1.00*** |

*Note.* The post-hoc power analyses for the interaction effects of the single moderation models were computed with GPower (Faul, Erdfelder, Lang, & Buchner, 2007), with the following specifications: model = linear multiple regression model $R^2$ increase, analysis = post-hoc: compute achieved power, N = 1130, α = 0.05, N of predictors = 3 (2 main effects, 1 interaction effect), tested predictors = 1, we then used the increase in $R^2$ after taking the interaction effect into account to calculate the $f^2$ effect size for the interaction effect. The post-hoc power analyses for the indirect effect of the single mediation models were conducted with the power analysis app written by Schoemann, Boulton, & Short (2017), with the following specifications: N = 1130, 1000 replications, 100000 Monte Carlo Draws per replication, seed = 4444, 95% CI, and specified the CA-RF, CA-distress, and RF-distress correlations (alongside the standard deviations of the three variables) . *** power ≥ 0.80, ** power ≥ 0.65, * power ≥ 0.50

## APPENDIX E.4: TESTING MULTICOLLINEARITY

For the models without gender and age-14 distress, none of the RFs had a variance inflation factor (VIF) above 3, regardless of investigating the CA+, the CA- or the whole sample (see Table E.5). For the models with gender, but without age-14 distress, none of the RFs had a VIF above 3, regardless of investigating the CA+, the CA- or the whole sample. For the models with gender and age-14 distress, negative self-esteem had a VIF above 3 in the whole sample as well as in the CA+ group. Yet, negative self-esteem had a VIF lower than 3 in the CA- group. Moreover, for the models with gender and age-14 distress, age-14 distress had a VIF above 3 in all three samples (i.e. the whole sample, the CA+ and the CA- group). Importantly, when taking the square root of the VIFs, none was bigger than 2, which indicates that even those variables that have a VIF above 3, are only borderline cases for multicollinearity.

Table E.5
*Variance inflation factors*

| | CA | gender | Frn | Fms | Fmc | Pst | Ngt | Brd | Rfl | Dst | Agg | Exp | D14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| WS | 1.08 | - | 1.22 | 1.90 | 2.11 | 1.82 | 2.40 | 2.27 | 1.66 | 1.15 | 1.13 | 1.06 | - |
| CA+ | - | - | 1.21 | 1.95 | 2.13 | 1.83 | 2.54 | 2.43 | 1.80 | 1.16 | 1.11 | 1.07 | - |
| CA- | - | - | 1.29 | 1.82 | 1.90 | 1.72 | 2.20 | 2.09 | 1.54 | 1.10 | 1.15 | 1.06 | - |
| WS | 1.08 | 1.23 | 1.23 | 1.91 | 2.11 | 1.85 | 2.41 | 2.27 | 1.75 | 1.17 | 1.17 | 1.06 | - |
| CA+ | - | 1.18 | 1.22 | 1.95 | 2.14 | 1.85 | 2.56 | 2.43 | 1.84 | 1.17 | 1.16 | 1.08 | - |
| CA- | - | 1.33 | 1.30 | 1.83 | 1.90 | 1.76 | 2.20 | 2.09 | 1.71 | 1.13 | 1.19 | 1.07 | - |
| WS | 1.08 | 1.24 | 1.27 | 1.91 | 2.13 | 1.87 | 3.03 | 2.49 | 1.76 | 1.17 | 1.21 | 1.06 | 3.52 |
| CA+ | - | 1.20 | 1.29 | 1.95 | 2.17 | 1.88 | 3.23 | 2.62 | 1.85 | 1.18 | 1.21 | 1.08 | 3.79 |
| CA- | - | 1.33 | 1.32 | 1.83 | 1.92 | 1.78 | 2.74 | 2.34 | 1.73 | 1.13 | 1.21 | 1.07 | 3.13 |

*Note.* WS = whole sample, CA+ = with childhood adversity, CA- = without childhood adversity; Frn = friendship support; Fms = family support; Fmc = family cohesion; Ngt = negative self-esteem; Pst = positive self-esteem; Brd = brooding; Rfl = reflection; Dst = distress tolerance; Agg = aggression; Exp = expressive suppression; D14 = age-14 distress. When taking the square root of the variance inflation factors, none was bigger than 2, which additionally underpins the absence of multicollinearity.

## APPENDIX E.5: RESULTS FOR MULTIPLE MODERATION AND MEDIATION EFFECTS, WITH AND WITHOUT CORRECTION FOR GENDER

This appendix contains the multiple moderation and mediation results, with and without correction for gender. The right panel of Table E.6 corresponds to Figure 3 in the Chapter. The right panel of Table E.7 corresponds to Figure 3 in the Chapter.

Table E.6
*Multiple moderation effects of the RFs without and with correction for gender*

| | ***Without gender*** | | | ***With gender*** | | |
|---|---|---|---|---|---|---|
| | b | SE | p | b | SE | p |
| CA | 0.27 | .07 | **<0.001*** | 0.26 | .07 | **<0.001*** |
| Friendships | 0.04 | .08 | 0.58 | 0.04 | .08 | 0.65 |
| Family support | -0.14 | .08 | **0.10** | -0.14 | .08 | **0.08** |
| Family cohesion | 0.02 | .09 | 0.80 | 0.03 | .09 | 0.78 |
| Positive self-esteem | -0.15 | .08 | **0.06** | -0.14 | .08 | **0.08** |
| Negative self-esteem | -0.41 | .10 | **<0.001*** | -0.41 | .10 | **<0.001*** |
| Brooding | -0.18 | .09 | **0.05** | -0.18 | .09 | **0.05** |
| Reflection | -0.09 | .08 | 0.28 | -0.06 | .08 | 0.45 |
| Distress tolerance | 0.01 | .06 | 0.83 | 0.02 | .06 | 0.73 |
| Aggression | -0.06 | .07 | 0.38 | -0.07 | .07 | 0.31 |
| Expressive suppression | 0.05 | .06 | 0.34 | 0.05 | .06 | 0.40 |
| Gender | | | | -0.11 | .08 | 0.17 |
| CA*Friendships | -0.19 | .10 | **0.06** | -0.19 | .10 | **0.07** |
| CA*Family support | 0.15 | .11 | 0.17 | 0.16 | .11 | 0.15 |
| CA*Family cohesion | -0.19 | .12 | 0.10 | -0.20 | .12 | **0.09** |
| CA*Positive self-esteem | -0.04 | .10 | 0.72 | -0.04 | .10 | 0.70 |
| CA*Negative self-esteem | 0.35 | .14 | <**0.05*** | 0.35 | .14 | **<0.05*** |
| CA*Brooding | 0.00 | .12 | 0.97 | 0.01 | .12 | 0.96 |
| CA*Reflection | 0.09 | .11 | 0.39 | 0.08 | .11 | 0.45 |
| CA*Distress tolerance | -0.14 | .08 | **0.10** | -0.14 | .08 | **0.09** |
| CA*Aggression | -0.07 | .08 | 0.36 | -0.07 | .08 | 0.35 |
| CA*Expressive Suppression | 0.00 | .07 | 0.99 | 0.01 | .07 | 0.94 |
| *% $R^2$* | | | 21% | | | 21% |

*Note.* b = unstandardized regression (slope) coefficient, SE = standard error, p = p-value, % $R^2$ = percentage variance explained.CA = childhood adversity,

Table E.7
*Multiple mediation effects of the RFs without and with correction for gender*

| | | ***Without gender*** | | | ***With gender*** | | |
|---|---|---|---|---|---|---|---|
| Path | | EST | p | % $R^2$ | EST | p | % $R^2$ |
| CA[1] → age17-distress | c | 0.44 | **<0.001*** | | 0.43 | **<0.001*** | |
| CA → age17-distress | c' | 0.25 | **<0.01*** | | 0.25 | **<0.01*** | |
| Friendships → age17-distress | b1 | -0.07 | 0.17 | | -0.07 | 0.14 | |
| Family sup. → age17-distress | b2 | -0.05 | 0.41 | | -0.05 | 0.37 | |
| Family coh. → age17-distress | b3 | -0.09 | **0.10** | | -0.09 | **0.09** | |
| Pos. SE → age17-distress | b4 | -0.17 | **<0.01*** | | -0.16 | **<0.01*** | |
| Neg. SE → age17-distress | b5 | -0.21 | **<0.01*** | | -0.20 | **<0.01*** | |
| Brooding → age17-distress | b6 | -0.18 | **<0.01*** | | -0.18 | **<0.01*** | |
| Reflection → age17-distress | b7 | -0.02 | 0.76 | | 0.00 | 0.98 | |
| Distress tol. → age17-distress | b8 | -0.06 | 0.14 | | -0.05 | 0.20 | |
| Aggression → age17-distress | b9 | -0.10 | **<0.05*** | | -0.11 | **<0.01*** | |
| Exp. sup. → age17-distress | b10 | 0.06 | **0.08** | | 0.06 | **0.09** | |
| Gender → age17-distress | cov | - | - | | -0.11 | 0.18 | |
| CA[1] → Friendships | a1 | -0.13 | **<0.01*** | 0.7 | -0.13 | **<0.01*** | 0.7 |
| CA[1] → Family sup. | a2 | -0.16 | **<0.01*** | 0.9 | -0.17 | **<0.01*** | 0.9 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| CA[1] → Family coh. | a3 | -0.36 | **<0.001*** | 4.3 | -0.36 | **<0.001*** | 4.4 |
| CA[1] → Pos. SE | a4 | -0.28 | **<0.001*** | 2.3 | -0.27 | **<0.001*** | 6.3 |
| CA[1] → Neg. SE | a5 | -0.19 | **<0.001*** | 1.4 | -0.18 | **<0.001*** | 5.5 |
| CA[1] → Brooding | a6 | -0.15 | **<0.01*** | 0.8 | -0.14 | **<0.01*** | 5.8 |
| CA[1] → Reflection | a7 | -0.07 | 0.19 | 0.2 | -0.04 | 0.36 | 10.0 |
| CA[1] → Distress tol. | a8 | -0.26 | **<0.001*** | 2.0 | -0.24 | **<0.001*** | 6.0 |
| CA[1] → Aggression | a9 | -0.23 | **<0.001*** | 1.3 | -0.24 | **<0.001*** | 3.2 |
| CA[1] → Exp. sup. | a10 | -0.18 | **<0.01*** | 0.8 | -0.18 | **<0.01*** | 1.5 |
| indirect effect friendships | a1*b1 | 0.009 | 0.22 | | 0.010 | 0.20 | |
| indirect effect family sup. | a2*b2 | 0.008 | 0.44 | | 0.008 | 0.40 | |
| indirect effect family coh. | a3*b3 | 0.034 | 0.11 | | 0.034 | 0.10 | |
| indirect effect pos. SE | a4*b4 | 0.048 | **<0.01*** | | 0.043 | **<0.01*** | |
| indirect effect neg. SE | a5*b5 | 0.039 | **<0.05*** | | 0.035 | **<0.05*** | |
| indirect effect brooding | a6*b6 | 0.027 | **<0.05*** | | 0.024 | **0.06** | |
| indirect effect reflection | a7*b7 | 0.001 | 0.81 | | -0.000 | 0.98 | |
| indirect effect distress tol. | a8*b8 | 0.015 | 0.15 | | 0.013 | 0.21 | |
| indirect effect aggression | a9*b9 | 0.023 | **<0.05*** | | 0.026 | **<0.05*** | |
| indirect effect exp. sup. | a10*b10 | -0.011 | 0.15 | | -0.011 | 0.15 | |
| Total indirect effect | ind | 0.193 | **<0.001*** | | 0.183 | **<0.001*** | |
| Total indirect RF effect | ind RF | 0.204 | **<0.001*** | | 0.194 | **<0.001*** | |
| Total effect | c'+ind | 0.441 | **<0.001*** | 20.0 | 0.429 | **<0.001*** | 20.0 |

*Note.* c = direct effect (that is the effect of CA alone, and if included gender). c' = corrected direct effect (that is the effect of CA, after taking into account all RFs, and if included gender). % $R^2$ = percentage variance explained. [1] = This path is in the second model also controlled for gender. Sup. = support, coh. = cohesion, SE = self-esteem, pos. = positive, neg. = negative, tol. = tolerance, Exp. sup. = Expressive suppression, cov = covariate. Total indirect effect = includes all 10 RFs. Total indirect RF effect = includes only those RFs that qualified as RF mediators (i.e. those with a negative a- and b-path). Total effect = that is the corrected direct effect of CA, plus the total indirect effect that goes via the RFs, and if included gender. Please note, the reported estimates and p-values are derived from the maximum likelihood models with bootstrapped standard errors.

# APPENDIX F

# Appendix Chapter 7

## APPENDIX F.1: PROOF OF PRINCIPAL ANALYSES FOR PERCEIVED STRESS AND MENTAL DISTRESS

Table F.1 depicts perceived stress and mental distress levels at the three occasions for all participants who took part in the respective occasion.

Table F.1
*Perceived stress and mental distress levels for the three occasions*

| | Occasion 1 M(SD), N | Occasion 2 M(SD), N | Occasion 3 M(SD), N |
|---|---|---|---|
| **Perceived stress** | 10.42(2.77), 451 | 11.61(2.77), 274 | 9.89(2.67), 282 |
| **Mental distress** | 25.40(5.82), 445 | 27.39(6.09), 273 | 23.31(5.93), 282 |

*Note.* M = mean, SD = standard deviation, N = sample size. Perceived stress was measured with the four-item Perceived Stress Scale (PSS; S. Cohen, Kamarck, & Mermelstein, 1983), details can be found in the RESIST protocol manuscript (J. Fritz, Stochl, Kievit, van Harmelen, & Wilkinson, 2020).

### F.1.1 Latent Growth Models (LGMs)

**Perceived stress.** The LGM showed that, on average, students experience most perceived stress during exams (occasion 2, slope loading fixed to 1), least perceived stress after the exams (occasion 3, slope loading fixed to 0), and before the exams they experienced more perceived stress than after the exams, but less than during the exams (occasion 1, estimated slope loading = 0.29; see Table F.2). The mean level trajectory is depicted in Figure F.1, left panel. We further found that the model estimating the change trajectory of perceived stress fits significantly better than the “no-change” model (see Table F.3), indicating that perceived stress changed significantly over the three occasions.

**Mental distress.** The LGM showed that, on average, students experience most mental distress during exams (occasion 2, slope loading fixed to 1), least mental distress after exams (occasion 3, slope loading fixed to 0), and before the exams they experienced more mental distress than after the exams, but less than during the exams (occasion 1, estimated slope loading = 0.60; see Table F.2). The mean level trajectory is depicted in Figure F.1, right panel. We further found that the model estimating the change trajectory of mental distress fits significantly better than the “no-change” model (see Table F.3), indicating that mental distress changed significantly over the three occasions.

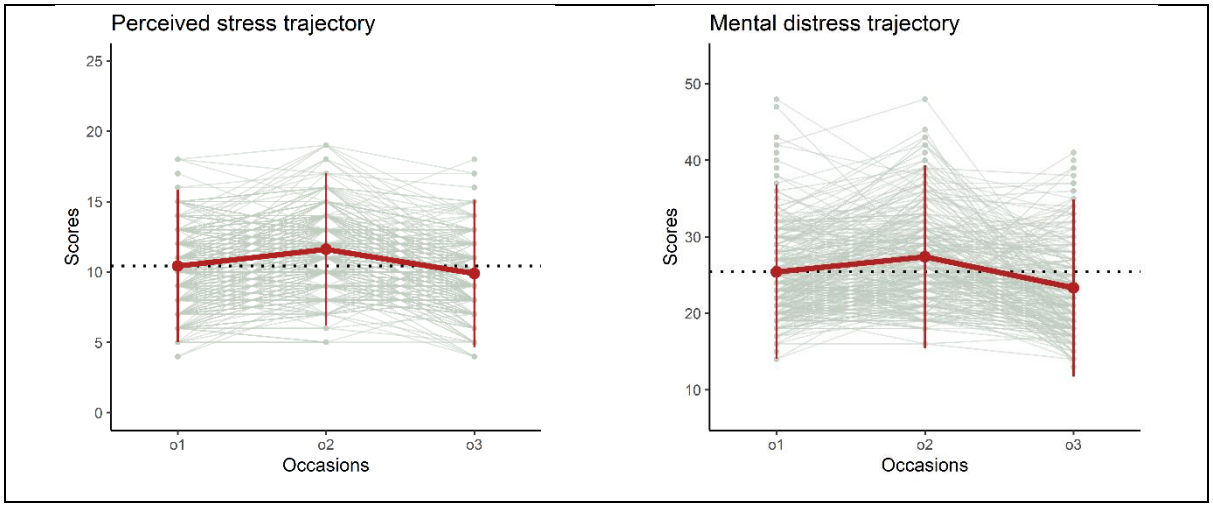



*Figure F.1.* The left panel depicts the perceived stress (sum-score mean-level) trajectory and the right panel the mental distress (sum-score mean-level) trajectory. The faded grey lines indicate person-level trajectories. The red line indicates the group-level sum-score trajectory. And the dotted black line represents the group-level sum-score for occasion 1, solely to enhance the comparison with the other occasions.

Table F.2
*Latent growth model summary*

| Model | Slope ld. o1 | Slope ld. o2 | Slope ld. o3 | Intercept mean | Slope mean | Residual var. o1 | Residual var. o2 | Residual var. o3 | Intercept slope cv. |
|---|---|---|---|---|---|---|---|---|---|
| **Perceived stress** | | | | | | | | | |
| M1 | 0.29 | 1.00 | 0.00 | 09.92 | 01.79 | 3.78 | 3.78 | 3.78 | 0.45 |
| M2 | 0.52 | 1.00 | 0.00 | 10.54 | 00.00 | 3.68 | 3.68 | 3.68 | -1.35 |
| M1* | 0.29 | 1.00 | 0.00 | 09.91 | 01.79 | 3.75 | 3.75 | 3.75 | 0.41 |
| M2* | 0.74 | 1.00 | 0.00 | 10.55 | 00.00 | 4.55 | 4.55 | 4.55 | 0.49 |
| M1** | 0.29 | 1.00 | 0.00 | 09.91 | 01.79 | 3.75 | 3.75 | 3.75 | 0.41 |
| M2** | 0.52 | 1.00 | 0.00 | 10.54 | 00.00 | 3.68 | 3.68 | 3.68 | -1.35 |
| **Mental distress** | | | | | | | | | |
| M1 | 0.60 | 1.00 | 0.00 | 23.21 | 04.08 | 16.85 | 16.85 | 16.85 | -8.71 |
| M2 | -4.41 | 1.00 | 0.00 | 25.44 | 00.00 | 37.50 | 37.50 | 37.50 | -1.65 |
| M1* | 0.48 | 1.00 | 0.00 | 23.39 | 04.18 | 22.20 | 22.20 | 22.20 | 2.96 |
| M2* | 0.38 | 1.00 | 0.00 | 25.26 | 00.00 | 26.50 | 26.50 | 26.50 | 2.60 |
| M1** | 0.60 | 1.00 | 0.00 | 23.21 | 04.08 | 16.85 | 16.85 | 16.85 | -8.71 |
| M2** | 0.67 | 1.00 | 0.00 | 25.26 | 00.00 | 16.74 | 16.74 | 16.74 | -18.07 |

*Note.* M1 = the freely estimated trajectory model. M2 = the no-change model. ld. = loading, o = occasion, var. = variance, cv. = covariance. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative.

Table F.3
*Latent growth model fit*

| Model | AIC | BIC | CFI | TLI | RMSEA | SRMR | $Chi^2$ (df) | BICw | AICw |
|---|---|---|---|---|---|---|---|---|---|
| **Perceived stress** | | | | | | | | | |
| M1 | 4718.05 | 4746.83 | 0.98 | 0.96 | 0.07 | 0.04 | 6.46 (2) | 100.00% | 100.00% |
| M2 | 4811.92 | 4836.59 | 0.47 | 0.47 | 0.27 | 0.18 | 102.33 (3) | 00.00% | 00.00% |
| M1* | 4716.06 | 4740.73 | 0.98 | 0.98 | 0.05 | 0.04 | 6.47 (3) | 100.00% | 100.00% |
| M2* | 4821.37 | 4841.93 | 0.41 | 0.56 | 0.25 | 0.17 | 113.78 (4) | 00.00% | 00.00% |
| M1** | 4718.06 | 4746.84 | 0.98 | 0.96 | 0.07 | 0.04 | 6.47 (2) | 100.00% | 100.00% |
| M2** | 4811.92 | 4836.59 | 0.47 | 0.47 | 0.27 | 0.18 | 102.33 (3) | 00.00% | 00.00% |
| M1 vs M2 | | $Chi^2$ difference = 72.37, df = 1, $p < 0.001$ | | | | | | | |
| M1* vs M2* | | $Chi^2$ difference = 97.44, df = 1, $p < 0.001$ | | | | | | | |
| M1** vs M2** | | $Chi^2$ difference = NC, df = 1, $p$ = NC | | | | | | | |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Mental distress** | | | | | | | | | |
| M1 | 6295.92 | 6324.66 | 0.94 | 0.91 | 0.09 | 0.05 | 9.18 (2) | 100.00% | 100.00% |
| M2 | 6366.44 | 6391.08 | 0.32 | 0.32 | 0.24 | 0.19 | 81.71 (3) | 00.00% | 00.00% |
| M1* | 6309.54 | 6334.18 | 0.81 | 0.81 | 0.13 | 0.07 | 24.81 (3) | 100.00% | 100.00% |
| M2* | 6403.08 | 6423.61 | 0.00 | 0.25 | 0.26 | 0.19 | 120.34 (4) | 00.00% | 00.00% |
| M1** | 6295.92 | 6324.66 | 0.94 | 0.91 | 0.09 | 0.05 | 9.18 (2) | 100.00% | 100.00% |
| M2** | 6364.52 | 6389.17 | 0.34 | 0.34 | 0.24 | 0.18 | 79.79 (3) | 00.00% | 00.00% |
| M1 vs M2 | | $Chi^2$ difference = 63.51, df = 1, $p < 0.001$ | | | | | | | |
| M1* vs M2* | | $Chi^2$ difference = 92.77, df = 1, $p < 0.001$ | | | | | | | |
| M1** vs M2** | | $Chi^2$ difference = 58.77, df = 1, $p < 0.001$ | | | | | | | |

*Note.* M1 = the freely estimated trajectory model. M2 = the no-change model. AIC = Akaike information criterion, BIC = Bayesian information criterion, CFI = confirmatory fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, df = degrees of freedom, p = p-value. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative. NC = could not be computed as the scaling factor was negative or the model did not converge.

### F.1.2 Bivariate Latent Change Score Models (BLCSMs)

**Occasion 1 to 2.** The BLCSM showed that perceived stress and mental distress at occasion 1 are positively associated, which indicates that students with higher perceived stress report on average higher mental distress (see Figure F.2, upper left panel). Similarly, change in perceived stress and mental distress from occasion 1 to 2 are positively associated, which indicates that students with more increase in perceived stress report on average also more increase in mental distress. Perceived stress was not associated with change in mental distress from occasion 1 to 2. However, mental distress was significantly positively associated with change in perceived stress from occasion 1 to 2, which indicates that higher mental distress at occasion 1 results on average in more increase in perceived stress from occasion 1 to 2.

**Occasion 1 to 3.** The BLCSM showed that changes in perceived stress and mental distress from occasion 1 to 3 are positively associated, which indicates that individuals with more decrease in perceived stress report on average also more decrease in mental distress (see Figure F.2, upper right panel). Mental distress was not associated with change in perceived stress from occasion 1 to 3. Perceived stress was positively associated with change in mental distress from occasion 1 to 3, yet, the p-value was only marginal (beta = .15, p = .06). The result may potentially indicate that higher perceived stress results on average in less decrease in mental distress from occasion 1 to 3, but needs to be interpreted with caution, or not at all, as the result was only marginal, not significant.

**Occasion 2 to 3.** The BLCSM showed that perceived stress and mental distress at occasion 2 are positively associated, which indicates that individuals with higher perceived stress report on average higher mental distress (see Figure F.2, lower left panel). Similarly, change in perceived stress and mental distress from occasion 2 to 3 are positively associated, which indicates that individuals with more decrease in perceived stress report on average also more decrease in mental distress. Mental distress was not associated with change in perceived stress from occasion 2 to 3. Yet, perceived stress was positively associated with change in mental distress from occasion 2 to 3, which indicates that higher perceived stress results on average in less decrease in mental distress from occasion 2 to 3.

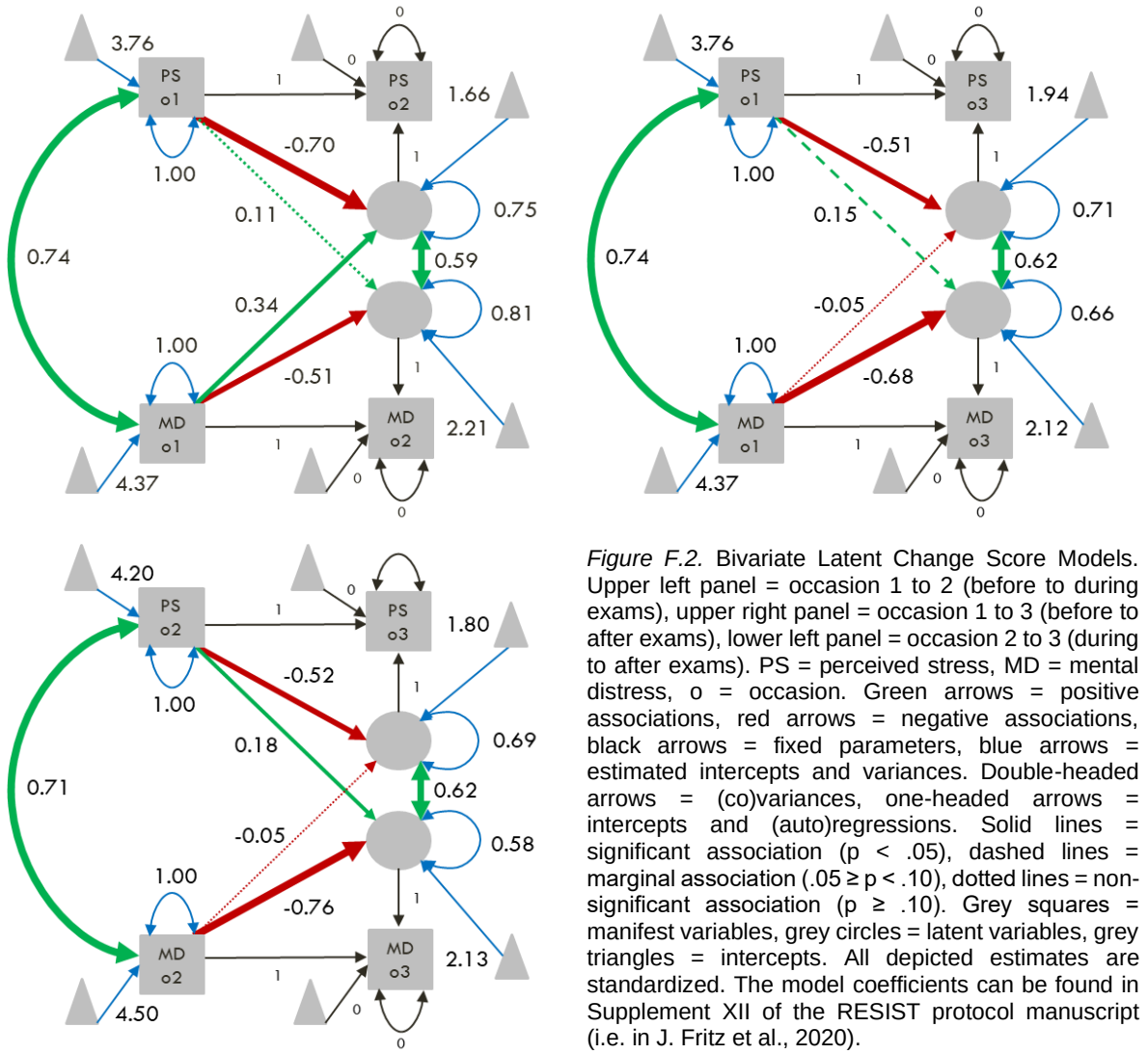



*Figure F.2.* Bivariate Latent Change Score Models. Upper left panel = occasion 1 to 2 (before to during exams), upper right panel = occasion 1 to 3 (before to after exams), lower left panel = occasion 2 to 3 (during to after exams). PS = perceived stress, MD = mental distress, o = occasion. Green arrows = positive associations, red arrows = negative associations, black arrows = fixed parameters, blue arrows = estimated intercepts and variances. Double-headed arrows = (co)variances, one-headed arrows = intercepts and (auto)regressions. Solid lines = significant association ($p < .05$), dashed lines = marginal association ($.05 \geq p < .10$), dotted lines = non-significant association ($p \geq .10$). Grey squares = manifest variables, grey circles = latent variables, grey triangles = intercepts. All depicted estimates are standardized. The model coefficients can be found in Supplement XII of the RESIST protocol manuscript (i.e. in J. Fritz et al., 2020).

### F.1.3 Conclusion

Both perceived stress and mental distress are lower before the exams, i.e. during the regular University term, than during the exam period, but higher before the exams than after the exams. Higher mental distress during term time was on average associated with a larger increase in perceived stress from the term time to the exam period. Hence, students who already had mental health problems prior to the exam period, were most prone to develop increased levels of stress during the exam period. Higher perceived stress during term time, which potentially could indicate enduring or chronic stress, was on average marginally associated with less recovery in mental distress from term time until the post-exam time. That is, students who were already stressed in the period before the exams were marginally less successful (or quick) in recovering from mental distress after the exams. Higher perceived stress during the exam period was on average associated with significantly less recovery in mental distress after the exam time. Thus, students who reported high stress during the exam period, were less successful (or quick) in recovering from mental distress. All in all, (a) persistent stress prior to exams may over time result in less recovery of mental distress after exams, (b) mental health problems prior to the exams

increase the risk for higher perceived stress during exams, and (c) higher perceived stress during exams in turn increases the risk for a less successful (or quick) recovery of mental distress after exams.

## APPENDIX F.2: RESULTS WITH AUXILIARIES

Results (see Tables F.5 to F.9) conducted with the missingness predictors (see Table F.4): Global stress severity at occasion 1 (i.e. 100-slider), medication use at occasion 1, gender and ethnicity.

Table F.4
*Examining potential missingness predictors for the analyses variables*

| | **Brooding** | | | **Self-Esteem** | | | **Mental Distress** | | |
|---|---|---|---|---|---|---|---|---|---|
| | **o1** | **o2** | **o3** | **o1** | **o2** | **o3** | **o1** | **o2** | **o3** |
| Missing responses | 001 | 176 | 168 | 006 | 180 | 173 | 006 | 178 | 169 |
| Brooding o1 | - | - | - | - | 0.74 | 0.86 | - | 0.94 | 0.84 |
| Brooding o2 | - | - | - | - | 0.12 | 0.79 | - | 0.90 | 0.87 |
| Brooding o3 | - | - | - | - | 0.38 | 0.33 | - | 0.52 | - |
| Self-esteem o1 | - | 0.98 | 0.69 | - | - | - | - | 1 | 0.78 |
| Self-esteem o2 | - | - | 0.53 | - | - | - | - | 0.83 | 0.42 |
| Self-esteem o3 | - | 0.27 | - | - | - | - | - | 0.25 | - |
| Perceived stress o1 | - | 0.12 | 0.32 | - | 0.11 | 0.39 | - | 0.12 | 0.25 |
| Perceived stress o2 | - | - | 0.41 | - | 0.57 | 0.40 | - | 0.63 | 0.44 |
| Perceived stress o3 | - | 0.91 | - | - | 0.94 | 0.54 | - | 0.83 | - |
| Global stress o1 | - | **<0.05*** | 0.33 | - | **<0.05*** | 0.49 | - | **<0.05*** | 0.26 |
| Global stress o2 | - | - | 0.78 | - | 0.18 | 0.60 | - | 0.58 | 0.78 |
| Global stress o3 | - | 0.14 | - | - | **0.07†** | 0.27 | - | 0.10 | - |
| Mental distress o1 | - | 0.14 | 0.46 | - | 0.13 | 0.47 | - | 0.12 | 0.38 |
| Mental distress o2 | - | - | 0.99 | - | 0.98 | 0.85 | - | - | 0.85 |
| Mental distress o3 | - | 0.45 | - | - | 0.67 | 0.99 | - | 0.43 | - |
| Psychotherapy o1 | - | 0.82 | 0.29 | - | 0.92 | 0.34 | - | 1 | 0.50 |
| Psychotherapy o2 | - | - | **0.08†** | - | 0.33 | 0.17 | - | - | 0.33 |
| Psychotherapy o3 | - | 0.18 | - | - | **0.10†** | - | - | 0.13 | - |
| Medication use o1 | - | **<0.05*** | **0.05†** | - | **0.09†** | **<0.05*** | - | **<0.05*** | 0.13 |
| Medication use o2 | - | - | 0.45 | - | **<0.001*** | 0.33 | - | - | 0.94 |
| Medication use o3 | - | 0.26 | - | - | 0.98 | - | - | 0.32 | - |
| Gender | - | **0.06†** | **0.08†** | - | **0.08†** | 0.12 | - | **<0.05*** | **0.09†** |
| Ethnicity | - | **<0.01*** | **0.05†** | - | **<0.01*** | **0.09†** | - | **<0.01*** | **<0.05*** |
| Academic year | - | 0.89 | 0.31 | - | 0.81 | 0.13 | - | 0.83 | 0.25 |
| Age | - | 0.98 | 0.44 | - | 0.83 | 0.25 | - | 0.92 | 0.42 |
| Retro. reported stress | - | 0.15 | - | - | 0.17 | 0.54 | - | 0.19 | - |
| Adversity | - | 0.31 | 0.96 | - | 0.30 | 0.91 | - | 0.38 | 0.85 |
| Adversity (binary) | - | 0.12 | 0.41 | - | 0.13 | 0.31 | - | 0.17 | 0.38 |

*Note.* O = occasion. Retro. reported stress = Retrospective (o3) reported exam stress. Missing responses counts the number of missing responses out of the 457 possible responses. All other depicted values represent p values that describe the (non)significance with which the variables predict missingness of the analyses variables. For binary predictors we conducted Pearson's Chi-squared tests with Yates' continuity correction and for continuous predictors Wilcoxon rank sum tests with continuity correction. Some tests could not successfully be conducted, as there either was too little missingness or too much overlapping missingness, which is indicated with "-". All analyses variables can be considered continuous. All variables are described in detail in the Chapter and/or in the related protocol paper (see J. Fritz et al., 2020). Medication use is limited to psychopharmacological medication.

Table F.5
*Brooding and self-esteem levels for the three occasions*

| | Occasion 1 M(SD), N | Occasion 2 M(SD), N | Occasion 3 M(SD), N |
|---|---|---|---|
| **Brooding** | 14.22(3.25), 450 | 14.10(3.29), 275 | 14.27(3.30), 283 |
| **Self-esteem** | 47.34(12.13), 445 | 45.88(12.40), 271 | 47.54(11.25), 278 |

*Note.* M = mean, SD = standard deviation, N = sample size.

Table F.6
*Latent growth model fit*

| Model | AIC | BIC | CFI | TLI | RMSEA | SRMR | Chi$^2$ (df) | BICw | AICw |
|---|---|---|---|---|---|---|---|---|---|
| **Brooding** | | | | | | | | | |
| M1* | 6706.87 | 6838.44 | 1.00 | 1.00 | 0.00 | 0.01 | 1.656 (3) | 08.90% | 36.27% |
| M2* | 6706.87 | 6838.44 | 1.00 | 1.00 | 0.00 | 0.01 | 1.656 (3) | - | - |
| M3* | 6708.21 | 6839.77 | 1.00 | 1.00 | 0.00 | 0.01 | 2.991 (3) | 04.57% | 18.61% |
| M4* | 6706.44 | 6833.89 | 1.00 | 1.00 | 0.00 | 0.01 | 3.219 (4) | 86.53% | 45.12% |
| M1* vs M4* | | Chi$^2$ difference = 1.02, df = 1, $p$ = 0.31 | | | | | | | |
| **Self-esteem** | | | | | | | | | |
| M1* | NC | NC | NC | NC | NC | NC | NC | NC | NC |
| M2* | 8897.42 | 9028.99 | 0.98 | 0.98 | 0.11 | 0.02 | 19.131 (3) | 100% | 100% |
| M3* | 8917.36 | 9048.92 | 0.95 | 0.95 | 0.16 | 0.03 | 39.063 (3) | 0% | 0% |
| M4* | NC | NC | NC | NC | NC | NC | NC | NC | NC |
| M1* vs M4* | | Chi$^2$ difference = NP, df = NP, $p$ = *NP* | | | | | | | |
| **Self-esteem** | | | | | | | | | |
| M1** | 8892.01 | 9027.69 | 0.99 | 0.98 | 0.10 | 0.02 | 11.720 (2) | 88.25% | 98.19% |
| M2** | 8892.01 | 9027.69 | 0.99 | 0.98 | 0.10 | 0.02 | 11.721 (2) | - | - |
| M3** | 8904.95 | 9040.63 | 0.97 | 0.95 | 0.16 | 0.02 | 24.656 (2) | 00.14% | 00.15% |
| M4** | 8900.18 | 9031.75 | 0.97 | 0.97 | 0.12 | 0.02 | 21.887 (3) | 11.62% | 01.65% |
| M1** vs M4** | | Chi$^2$ difference = 6.34, df = 1, $p$ < 0.05 | | | | | | | |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. AIC = Akaike information criterion, BIC = Bayesian information criterion, CFI = confirmatory fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, df = degrees of freedom, p = p-value. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative. NC = not converged. NP = not possible due to model nonconvergence.

Table F.7
*Latent growth model summary*

| Model | Slope ld. o1 | Slope ld. o2 | Slope ld. o3 | Intercept mean | Slope mean | Residual var. o1 | Residual var. o2 | Residual var. o3 | Intercept slope cv. |
|---|---|---|---|---|---|---|---|---|---|
| **Brooding** | | | | | | | | | |
| M1* | 0.921 | 0.000 | 1.000 | 14.045 | 0.199 | 3.691 | 3.691 | 3.691 | -0.216 |
| M2* | 0.921 | 0.000 | 1.000 | 14.045 | 0.199 | 3.691 | 3.691 | 3.691 | -0.216 |
| M3* | 0.000 | 0.000 | 1.000 | 14.163 | 0.112 | 3.697 | 3.697 | 3.697 | 0.065 |
| M4* | 2.440 | 0.000 | 1.000 | 14.194 | 0.000 | 3.703 | 3.703 | 3.703 | -0.119 |
| **Self-esteem** | | | | | | | | | |
| M1* | NP1 | 0.000 | 1.000 | 46.509 | 0.000 | 25.058 | 25.058 | 25.058 | -0.000 |
| M2* | 0.806 | 0.000 | 1.000 | 45.608 | 1.977 | 24.332 | 24.332 | 24.332 | -10.187 |
| M3* | NP2 | 0.000 | 1.000 | 46.509 | 0.000 | 25.058 | 25.058 | 25.058 | -0.000 |
| M4* | NP3 | 0.000 | 1.000 | 46.863 | 0.000 | 25.308 | 25.308 | 25.308 | 0.000 |
| **Self-esteem** | | | | | | | | | |
| M1** | 0.429 | 0.000 | 1.000 | 46.199 | 1.658 | 19.499 | 19.499 | 19.499 | -25.766 |
| M2** | 0.429 | 0.000 | 1.000 | 46.199 | 1.658 | 19.495 | 19.495 | 19.495 | -25.818 |
| M3** | 0.000 | 0.000 | 1.000 | 46.774 | 0.784 | 20.661 | 20.661 | 20.661 | -17.575 |
| M4** | 0.320 | 0.000 | 1.000 | 47.198 | 0.000 | 19.229 | 19.229 | 19.229 | -26.838 |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. ld. = loading, o = occasion, var. = variance, cv. = covariance. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative. NP = not plausible, NP1 = -419652.64, NP2 = -72385.71, NP3 = 198576.29.

Table F.8
*Brooding: bivariate latent change score models*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression occasion 1 to 2** | | | | | |
| brooding o1 → distress c(o1-o2) | -0.280 | 0.128 | -0.160 | -2.182 | 0.029 |
| distress o1 → distress c(o1-o2) | -0.500 | 0.077 | -0.511 | -6.485 | 0.000 |
| distress o1 → brooding c(o1-o2) | -0.115 | 0.033 | -0.251 | -3.526 | 0.000 |
| brooding o1 → brooding c(o1-o2) | -0.434 | 0.053 | -0.528 | -8.186 | 0.000 |
| **(auto)regression occasion 1 to 3** | | | | | |
| brooding o1 → distress c(o1-o3) | -0.063 | 0.125 | -0.030 | -0.503 | 0.615 |
| distress o1 → distress c(o1-o3) | -0.708 | 0.078 | -0.594 | -9.056 | 0.000 |
| distress o1 → brooding c(o1-o3) | -0.009 | 0.032 | -0.018 | -0.272 | 0.786 |
| brooding o1 → brooding c(o1-o3) | -0.362 | 0.054 | -0.418 | -6.722 | 0.000 |
| **(auto)regression occasion 2 to 3** | | | | | |
| brooding o2 → distress c(o2-o3) | -0.216 | 0.128 | -0.096 | -1.691 | 0.091 |
| distress o2 → distress c(o2-o3) | -0.822 | 0.081 | -0.678 | -10.194 | 0.000 |
| distress o2 → brooding c(o2-o3) | -0.006 | 0.035 | -0.013 | -0.167 | 0.867 |
| brooding o2 → brooding c(o2-o3) | -0.336 | 0.053 | -0.414 | -6.329 | 0.000 |

*Note.* SE = standard error. O = occasion.

Table F.9
*Self-esteem: bivariate latent change score models*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression occasion 1 to 2** | | | | | |
| self-esteem o1 → distress c(o1-o2) | -0.120 | 0.031 | -0.256 | -3.867 | 0.000 |
| distress o1 → distress c(o1-o2) | -0.586 | 0.076 | -0.595 | -7.662 | 0.000 |
| distress o1 → self-esteem c(o1-o2) | -0.100 | 0.089 | -0.093 | -1.121 | 0.262 |
| self-esteem o1 → self-esteem c(o1-o2) | -0.145 | 0.038 | -0.281 | -3.804 | 0.000 |
| **(auto)regression occasion 1 to 3** | | | | | |
| self-esteem o1 → distress c(o1-o3) | -0.063 | 0.042 | -0.110 | -1.513 | 0.130 |
| distress o1 → distress c(o1-o3) | -0.777 | 0.091 | -0.650 | -8.513 | 0.000 |
| distress o1 → self-esteem c(o1-o3) | 0.034 | 0.106 | 0.028 | 0.323 | 0.747 |
| self-esteem o1 → self-esteem c(o1-o3) | -0.213 | 0.048 | -0.366 | -4.442 | 0.000 |
| **(auto)regression occasion 2 to 3** | | | | | |
| self-esteem o2 → distress c(o2-o3) | -0.072 | 0.041 | -0.120 | -1.760 | 0.078 |
| distress o2 → distress c(o2-o3) | -0.848 | 0.087 | -0.705 | -9.708 | 0.000 |
| distress o2 → self-esteem c(o2-o3) | 0.150 | 0.106 | 0.118 | 1.411 | 0.158 |
| self-esteem o2 → self-esteem c(o2-o3) | -0.234 | 0.045 | -0.371 | -5.181 | 0.000 |

*Note.* SE = standard error. O = occasion.

## APPENDIX F.3: RESULTS BASED ON THE COMPLETE SAMPLE

Results (see Tables F.10 to F.14) for the sample with full data for all analysis variables, n = 218.

Table F.10
*Brooding and self-esteem levels for the three occasions*

| | Occasion 1 M(SD), N | Occasion 2 M(SD), N | Occasion 3 M(SD), N |
|---|---|---|---|
| **Brooding** | 14.36(3.20), 218 | 14.23(3.30), 218 | 14.42(3.24), 218 |
| **Self-esteem** | 47.61(12.04), 218 | 46.14(12.23), 218 | 48.23(10.94), 218 |

*Note.* M = mean, SD = standard deviation, N = sample size.

Table F.11
*Latent growth model fit*

| Model | AIC | BIC | CFI | TLI | RMSEA | SRMR | $Chi^2$ (df) | BICw | AICw |
|---|---|---|---|---|---|---|---|---|---|
| **Brooding** | | | | | | | | | |
| M1* | 3131.49 | 3151.80 | 1.00 | 1.01 | 0.00 | 0.01 | 1.257 (3) | 09.70% | 31.72% |
| M2* | 3131.49 | 3151.80 | 1.00 | 1.01 | 0.00 | 0.01 | 1.257 (3) | - | - |
| M3* | 3132.73 | 3153.04 | 1.00 | 1.00 | 0.00 | 0.03 | 2.496 (3) | 05.22% | 17.07% |
| M4* | 3130.53 | 3147.46 | 1.00 | 1.01 | 0.00 | 0.02 | 2.298 (4) | 85.08% | 51.21% |
| M1* vs M4* | | $Chi^2$ difference = 0.72, df = 1, $p$ = 0.40 | | | | | | | |
| **Self-esteem** | | | | | | | | | |
| M1* | 4544.02 | 4564.33 | 0.98 | 0.98 | 0.14 | 0.04 | 16.602 (3) | 94.75% | 94.76% |
| M2* | 4544.02 | 4564.33 | 0.98 | 0.98 | 0.14 | 0.04 | 16.602 (3) | - | - |
| M3* | 4549.81 | 4570.12 | 0.97 | 0.97 | 0.17 | 0.03 | 22.392 (3) | 05.24% | 05.24% |
| M4* | 4565.45 | 4582.38 | 0.94 | 0.95 | 0.20 | 0.08 | 40.037 (4) | 00.01% | 00.00% |
| M1* vs M4* | | $Chi^2$ difference = 10.64, df = 1, $p$ < 0.01 | | | | | | | |
| **Self-esteem** | | | | | | | | | |
| M1** | 4535.14 | 4558.83 | 0.99 | 0.99 | 0.09 | 0.03 | 05.722 (2) | 97.51% | 98.89% |
| M2** | 4535.14 | 4558.83 | 0.99 | 0.99 | 0.09 | 0.03 | 05.722 (2) | - | - |
| M3** | 4544.81 | 4568.50 | 0.98 | 0.96 | 0.18 | 0.03 | 15.390 (2) | 00.78% | 00.79% |
| M4** | 4546.61 | 4566.91 | 0.97 | 0.97 | 0.16 | 0.05 | 19.189 (3) | 01.71% | 00.32% |
| M1* vs M4* | | $Chi^2$ difference = 13.45, df = 1, $p$ < 0.001 | | | | | | | |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. AIC = Akaike information criterion, BIC = Bayesian information criterion, CFI = confirmatory fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, df = degrees of freedom, p = p-value. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative.

Table F.12
*Latent growth model summary*

| Model | Slope ld. o1 | Slope ld. o2 | Slope ld. o3 | Intercept mean | Slope mean | Residual var. o1 | Residual var. o2 | Residual var. o3 | Intercept slope cv. |
|---|---|---|---|---|---|---|---|---|---|
| **Brooding** | | | | | | | | | |
| M1* | 0.942 | 0.000 | 1.000 | 14.223 | 0.175 | 3.703 | 3.703 | 3.703 | -0.429 |
| M2* | 0.942 | 0.000 | 1.000 | 14.223 | 0.175 | 3.703 | 3.703 | 3.703 | -0.429 |
| M3* | 0.000 | 0.000 | 1.000 | 14.294 | 0.128 | 3.707 | 3.707 | 3.707 | -0.088 |
| M4* | 1.350 | 0.000 | 1.000 | 14.348 | 0.000 | 3.713 | 3.713 | 3.713 | -0.360 |
| **Self-esteem** | | | | | | | | | |
| M1* | 0.526 | 0.000 | 1.000 | 46.259 | 2.104 | 24.757 | 24.757 | 24.757 | -13.466 |
| M2* | 0.526 | 0.000 | 1.000 | 46.259 | 2.104 | 24.757 | 24.757 | 24.757 | -13.466 |
| M3* | 0.000 | 0.000 | 1.000 | 46.876 | 1.358 | 25.252 | 25.252 | 25.252 | -13.794 |
| M4* | NP | 0.000 | 1.000 | 47.217 | 0.000 | 25.865 | 25.865 | 25.865 | -00.003 |

| **Self-esteem** | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| M1** | 0.383 | 0.000 | 1.000 | 46.411 | 1.990 | 19.230 | 19.230 | 19.230 | -27.549 |
| M2** | 0.383 | 0.000 | 1.000 | 46.411 | 1.990 | 19.230 | 19.230 | 19.230 | -27.549 |
| M3** | 0.000 | 0.000 | 1.000 | 46.876 | 1.358 | 20.760 | 20.760 | 20.760 | -20.757 |
| M4** | 0.307 | 0.000 | 1.000 | 47.898 | 0.000 | 19.235 | 19.235 | 19.235 | -29.664 |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. ld. = loading, o = occasion, var. = variance, cv. = covariance. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative. NP = not plausible = -3298.18.

Table F.13
*Brooding: bivariate latent change score models*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression occasion 1 to 2** | | | | | |
| brooding o1 → distress c(o1-o2) | -0.206 | 0.142 | -0.119 | -1.449 | 0.147 |
| distress o1 → distress c(o1-o2) | -0.462 | 0.080 | -0.463 | -5.802 | 0.000 |
| distress o1 → brooding c(o1-o2) | -0.132 | 0.037 | -0.272 | -3.556 | 0.000 |
| brooding o1 → brooding c(o1-o2) | -0.437 | 0.059 | -0.518 | -7.472 | 0.000 |
| **(auto)regression occasion 1 to 3** | | | | | |
| brooding o1 → distress c(o1-o3) | -0.071 | 0.135 | -0.033 | -0.530 | 0.596 |
| distress o1 → distress c(o1-o3) | -0.735 | 0.087 | -0.595 | -8.442 | 0.000 |
| distress o1 → brooding c(o1-o3) | -0.028 | 0.041 | -0.056 | -0.703 | 0.482 |
| brooding o1 → brooding c(o1-o3) | -0.401 | 0.064 | -0.456 | -6.311 | 0.000 |
| **(auto)regression occasion 2 to 3** | | | | | |
| brooding o2 → distress c(o2-o3) | -0.200 | 0.141 | -0.088 | -1.424 | 0.154 |
| distress o2 → distress c(o2-o3) | -0.857 | 0.082 | -0.693 | -10.460 | 0.000 |
| distress o2 → brooding c(o2-o3) | 0.001 | 0.035 | 0.002 | 0.026 | 0.980 |
| brooding o2 → brooding c(o2-o3) | -0.343 | 0.054 | -0.426 | -6.322 | 0.000 |

*Note.* SE = standard error. O = occasion.

Table F.14
*Self-esteem: bivariate latent change score models*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression occasion 1 to 2** | | | | | |
| self-esteem o1 → distress c(o1-o2) | -0.085 | 0.036 | -0.185 | -2.346 | 0.019 |
| distress o1 → distress c(o1-o2) | -0.525 | 0.082 | -0.527 | -6.407 | 0.000 |
| distress o1 → self-esteem c(o1-o2) | -0.191 | 0.095 | -0.169 | -2.000 | 0.046 |
| self-esteem o1 → self-esteem c(o1-o2) | -0.179 | 0.043 | -0.342 | -4.120 | 0.000 |
| **(auto)regression occasion 1 to 3** | | | | | |
| self-esteem o1 → distress c(o1-o3) | -0.040 | 0.044 | -0.070 | -0.893 | 0.372 |
| distress o1 → distress c(o1-o3) | -0.771 | 0.097 | -0.624 | -7.959 | 0.000 |
| distress o1 → self-esteem c(o1-o3) | 0.056 | 0.113 | 0.044 | 0.495 | 0.620 |
| self-esteem o1 → self-esteem c(o1-o3) | -0.240 | 0.052 | -0.412 | -4.654 | 0.000 |
| **(auto)regression occasion 2 to 3** | | | | | |
| self-esteem o2 → distress c(o2-o3) | -0.066 | 0.044 | -0.107 | -1.494 | 0.135 |
| distress o2 → distress c(o2-o3) | -0.888 | 0.091 | -0.718 | -9.815 | 0.000 |
| distress o2 → self-esteem c(o2-o3) | 0.204 | 0.106 | 0.160 | 1.926 | 0.054 |
| self-esteem o2 → self-esteem c(o2-o3) | -0.235 | 0.048 | -0.371 | -4.933 | 0.000 |

*Note.* SE = standard error. O = occasion.

## APPENDIX F.4: OVERVIEW OF USED R PACKAGES, INCLUDING THEIR VERSION NUMBER AND REFERENCE

| Package (version) | Reference |
|---|---|
| pastecs (1.3.21) | (Grosjean & Ibanez, 2018) |
| lavaan (0.6-4) | (Rosseel, 2012) |
| ggplot2 (3.1.0) | (Wickham, 2016) |
| reshape2 (1.4.3) | (Wickham, 2007) |
| semTools (0.5-1.933) | (Jorgensen et al., 2018) |
| dplyr (0.7.7) | (Wickham et al., 2018) |
| AICcmodavg (2.2-2) | (Mazerolle, 2019) |

## APPENDIX F.5: TESTING MULTICOLLINEARITY

*Variance inflation factors*

| Outcome | Predictor | | | |
|---|---|---|---|---|
| | **MD o1 & SE o1** | **MD o2 & SE o2** | **MD o1 & BRD o1** | **MD o2 & BRD o2** |
| **MD o2** | 1.77 | - | 1.38 | - |
| **MD o3** | 1.76 | 1.71 | 1.28 | 1.32 |
| **SE o2** | 1.77 | - | - | - |
| **SE o3** | 1.79 | 1.71 | - | - |
| **BRD o2** | - | - | 1.39 | - |
| **BRD o3** | - | - | 1.29 | 1.33 |

*Note.* MD = mental distress, SE = self-esteem, BRD = brooding, o = occasion.

## APPENDIX F.6: EXACT COEFFICIENTS FOR THE FIGURES PRESENTED IN CHAPTER 7

*Brooding and self-esteem levels for the three occasions.*

| | Occasion 1 M(SD), N | Occasion 2 M(SD), N | Occasion 3 M(SD), N |
|---|---|---|---|
| **Brooding** | 14.22(03.25), 450 | 14.10(03.29), 275 | 14.27(03.30), 283 |
| **Self-esteem** | 47.34(12.13), 445 | 45.88(12.40), 271 | 47.54(11.25), 278 |

*Note.* M = mean, SD = standard deviation, N = sample size.

*Robustness check for the self-esteem latent growth model fit*

| Model | AIC | BIC | CFI | TLI | RMSEA | SRMR | $Chi^2$ (df) | BICw | AICw |
|---|---|---|---|---|---|---|---|---|---|
| **Self-esteem** | | | | | | | | | |
| M1** | 7071.35 | 7100.06 | 0.99 | 0.98 | 0.10 | 0.03 | 11.425 (2) | 88.75% | 98.27% |
| M2** | 7071.35 | 7100.06 | 0.99 | 0.98 | 0.10 | 0.03 | 11.425 (2) | - | - |
| M3** | 7084.35 | 7113.06 | 0.97 | 0.95 | 0.16 | 0.04 | 24.423 (2) | 00.13% | 00.15% |
| M4** | 7079.60 | 7104.22 | 0.97 | 0.97 | 0.12 | 0.04 | 21.681 (3) | 11.12% | 01.58% |
| M1* vs M4* | | $Chi^2$ difference = 6.33, df = 1, $p < 0.05$ | | | | | | | |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. AIC = Akaike information criterion, BIC = Bayesian information criterion, CFI = confirmatory fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, df = degrees of freedom, p = p-value. **the variance for the latent slope constrained to >0, to render it non-negative.

*Robustness check for the self-esteem latent growth model summary*

| Model | Slope ld. o1 | Slope ld. o2 | Slope ld. o3 | Intercept mean | Slope mean | Residual var. o1 | Residual var. o2 | Residual var. o3 | Intercept slope cv. |
|---|---|---|---|---|---|---|---|---|---|
| **Self-esteem** | | | | | | | | | |
| M1** | 0.435 | 0.000 | 1.000 | 46.253 | 1.649 | 19.552 | 19.552 | 19.552 | -25.256 |
| M2** | 0.435 | 0.000 | 1.000 | 46.253 | 1.649 | 19.546 | 19.546 | 19.546 | -25.329 |
| M3** | 0.000 | 0.000 | 1.000 | 46.830 | 0.769 | 20.653 | 20.653 | 20.653 | -17.140 |
| M4** | 0.321 | 0.000 | 1.000 | 47.249 | 0.000 | 19.279 | 19.279 | 19.279 | -26.360 |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. ld. = loading, o = occasion, var. = variance, cv. = covariance. **the variance for the latent slope constrained to >0, to render it non-negative.

*Brooding: bivariate latent change score models summary: occasion 1 to 2*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression** | | | | | |
| brooding o1 → distress c(o1-o2) | -00.267 | 0.127 | -0.152 | -2.104 | 0.035 |
| distress o1 → distress c(o1-o2) | -00.499 | 0.078 | -0.510 | -6.402 | 0.000 |
| distress o1 → brooding c(o1-o2) | -00.119 | 0.033 | -0.260 | -3.619 | 0.000 |
| brooding o1 → brooding c(o1-o2) | -00.431 | 0.053 | -0.525 | -8.194 | 0.000 |
| **covariances** | | | | | |
| brooding o1 ←→ distress o1 | -10.168 | 1.045 | -00.540 | -9.730 | 0.000 |
| brooding c(o1-o2) ←→ distress c(o1-o2) | -03.313 | 0.760 | -00.273 | -4.358 | 0.000 |
| **intercepts** | | | | | |
| brooding c(o1-o2) | 08.998 | 1.376 | 03.381 | 06.541 | 0.000 |
| brooding o1 | 14.219 | 0.153 | 04.385 | 93.117 | 0.000 |
| distress c(o1-o2) | 18.647 | 3.318 | 03.278 | 05.620 | 0.000 |
| distress o1 | 25.381 | 0.274 | 04.370 | 92.533 | 0.000 |
| **variances** | | | | | |
| brooding c(o1-o2) | 05.695 | 0.475 | 00.804 | 11.996 | 0.000 |

| | | | | | |
|---|---|---|---|---|---|
| brooding o1 | 10.513 | 0.646 | 01.000 | 16.283 | 0.000 |
| distress c(o1-o2) | 25.914 | 2.242 | 00.801 | 11.557 | 0.000 |
| distress o1 | 33.725 | 2.644 | 01.000 | 12.755 | 0.000 |

*Note.* SE = standard error. O = occasion.

*Brooding: bivariate latent change score models summary: occasion 1 to 3*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression** | | | | | |
| brooding o1 → distress c(o1-o3) | -00.065 | 0.125 | -0.031 | -0.523 | 0.601 |
| distress o1 → distress c(o1-o3) | -00.702 | 0.077 | -0.591 | -9.118 | 0.000 |
| distress o1 → brooding c(o1-o3) | -00.014 | 0.032 | -0.029 | -0.437 | 0.662 |
| brooding o1 → brooding c(o1-o3) | -00.364 | 0.054 | -0.421 | -6.671 | 0.000 |
| **covariances** | | | | | |
| brooding o1 ←→ distress o1 | -10.169 | 1.044 | -0.540 | -9.737 | 0.000 |
| brooding c(o1-o3) ←→ distress c(o1-o3) | -03.709 | 1.016 | -0.257 | -3.650 | 0.000 |
| **intercepts** | | | | | |
| brooding c(o1-o3) | 05.568 | 1.405 | 1.987 | 3.964 | 0.000 |
| brooding o1 | 14.217 | 0.153 | 4.386 | 93.105 | 0.000 |
| distress c(o1-o3) | 16.755 | 3.241 | 2.430 | 5.170 | 0.000 |
| distress o1 | 25.384 | 0.274 | 4.372 | 92.514 | 0.000 |
| **variances** | | | | | |
| brooding c(o1-o3) | 06.561 | 0.599 | 0.836 | 10.947 | 0.000 |
| brooding o1 | 10.509 | 0.645 | 1.000 | 16.284 | 0.000 |
| distress c(o1-o3) | 31.818 | 2.831 | 0.669 | 11.241 | 0.000 |
| distress o1 | 33.716 | 2.644 | 1.000 | 12.753 | 0.000 |

*Note.* SE = standard error. O = occasion.

*Brooding: bivariate latent change score models summary: occasion 2 to 3*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression** | | | | | |
| brooding o2 → distress c(o2-o3) | -0.222 | 0.130 | -0.098 | -01.703 | 0.089 |
| distress o2 → distress c(o2-o3) | -0.833 | 0.081 | -0.683 | -10.274 | 0.000 |
| distress o2 → brooding c(o2-o3) | -0.003 | 0.035 | -0.008 | -00.101 | 0.920 |
| brooding o2 → brooding c(o2-o3) | -0.331 | 0.052 | -0.413 | -06.325 | 0.000 |
| **covariances** | | | | | |
| brooding o2 ←→ distress o2 | -9.908 | 1.308 | -0.492 | -07.575 | 0.000 |
| brooding c(o2-o3) ←→ distress c(o2-o3) | -3.087 | 0.958 | -0.224 | -03.224 | 0.001 |
| **intercepts** | | | | | |
| brooding c(o2-o3) | 4.971 | 1.444 | 1.881 | 03.442 | 0.001 |
| brooding o2 | 14.067 | 0.192 | 4.265 | 73.080 | 0.000 |
| distress c(o2-o3) | 21.861 | 3.485 | 2.935 | 06.273 | 0.000 |
| distress o2 | 27.439 | 0.366 | 4.495 | 74.992 | 0.000 |
| **variances** | | | | | |
| brooding c(o2-o3) | 5.816 | 0.600 | 0.832 | 09.694 | 0.000 |
| brooding o2 | 10.877 | 0.792 | 1.000 | 13.733 | 0.000 |
| distress c(o2-o3) | 32.727 | 2.939 | 0.590 | 11.136 | 0.000 |
| distress o2 | 37.256 | 3.166 | 1.000 | 11.768 | 0.000 |

*Note.* SE = standard error. O = occasion.

*Self-esteem: bivariate latent change score models summary: occasion 1 to 2*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression** | | | | | |
| self-esteem o1 → distress c(o1-o2) | -0.119 | 0.031 | -0.252 | -3.838 | 0.000 |
| distress o1 → distress c(o1-o2) | -0.587 | 0.077 | -0.596 | -7.604 | 0.000 |
| distress o1 → self-esteem c(o1-o2) | -0.107 | 0.090 | -0.099 | -1.192 | 0.233 |
| self-esteem o1 → self-esteem c(o1-o2) | -0.144 | 0.038 | -0.279 | -3.826 | 0.000 |
| **covariances** | | | | | |
| self-esteem o1 ←→ distress o1 | -46.355 | 3.699 | -0.658 | -12.531 | 0.000 |
| self-esteem c(o1-o2) ←→ distress c(o1-o2) | -17.638 | 2.174 | -0.573 | -08.114 | 0.000 |
| **intercepts** | | | | | |
| self-esteem c(o1-o2) | 07.924 | 3.651 | 1.266 | 02.171 | 0.030 |
| self-esteem o1 | 47.296 | 0.573 | 3.896 | 82.544 | 0.000 |
| distress c(o1-o2) | 22.681 | 3.060 | 3.967 | 07.413 | 0.000 |
| distress o1 | 25.372 | 0.274 | 4.370 | 92.616 | 0.000 |
| **variances** | | | | | |
| self-esteem c(o1-o2) | 037.199 | 3.305 | 0.949 | 11.254 | 0.000 |
| self-esteem o1 | 147.352 | 8.822 | 1.000 | 16.704 | 0.000 |
| distress c(o1-o2) | 025.462 | 2.200 | 0.779 | 11.575 | 0.000 |
| distress o1 | 033.704 | 2.645 | 1.000 | 12.741 | 0.000 |

*Note.* SE = standard error. O = occasion.

*Self-esteem: bivariate latent change score models summary: occasion 1 to 3*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression** | | | | | |
| self-esteem o1 → distress c(o1-o3) | -0.063 | 0.042 | -0.111 | -1.518 | 0.129 |
| distress o1 → distress c(o1-o3) | -0.772 | 0.090 | -0.648 | -8.572 | 0.000 |
| distress o1 → self-esteem c(o1-o3) | 0.026 | 0.105 | 0.021 | 0.244 | 0.807 |
| self-esteem o1 → self-esteem c(o1-o3) | -0.213 | 0.048 | -0.367 | -4.436 | 0.000 |
| **covariances** | | | | | |
| self-esteem o1 ←→ distress o1 | -46.593 | 3.692 | -0.661 | -12.619 | 0.000 |
| self-esteem c(o1-o3) ←→ distress c(o1-o3) | -18.935 | 2.873 | -0.516 | -06.590 | 0.000 |
| **intercepts** | | | | | |
| self-esteem c(o1-o3) | 09.593 | 4.559 | 1.363 | 02.104 | 0.035 |
| self-esteem o1 | 47.275 | 0.573 | 3.898 | 82.566 | 0.000 |
| distress c(o1-o3) | 20.605 | 3.895 | 2.976 | 05.290 | 0.000 |
| distress o1 | 25.373 | 0.274 | 4.368 | 92.575 | 0.000 |
| **variances** | | | | | |
| self-esteem c(o1-o3) | 042.347 | 4.148 | 0.855 | 10.208 | 0.000 |
| self-esteem o1 | 147.076 | 8.810 | 1.000 | 16.693 | 0.000 |
| distress c(o1-o3) | 031.791 | 2.804 | 0.663 | 11.340 | 0.000 |
| distress o1 | 033.743 | 2.648 | 1.000 | 12.742 | 0.000 |

*Note.* SE = standard error. O = occasion.

*Self-esteem: bivariate latent change score models summary: occasion 2 to 3*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression** | | | | | |
| self-esteem o2 → distress c(o2-o3) | -0.085 | 0.042 | -0.143 | -2.039 | 0.041 |
| distress o2 → distress c(o2-o3) | -0.874 | 0.089 | -0.722 | -9.810 | 0.000 |
| distress o2 → self-esteem c(o2-o3) | 0.172 | 0.105 | 0.135 | 1.635 | 0.102 |

| | | | | | |
|---|---|---|---|---|---|
| self-esteem o2 → self-esteem c(o2-o3) | -0.224 | 0.045 | -0.359 | -4.990 | 0.000 |
| **covariances** | | | | | |
| self-esteem o2 ←→ distress o2 | -50.686 | 5.266 | -0.668 | -9.625 | 0.000 |
| self-esteem c(o2-o3) ←→ distress c(o2-o3) | -19.549 | 3.354 | -0.497 | -5.828 | 0.000 |
| **intercepts** | | | | | |
| self-esteem c(o2-o3) | 07.382 | 4.393 | 0.951 | 01.680 | 0.093 |
| self-esteem o2 | 45.514 | 0.715 | 3.665 | 63.695 | 0.000 |
| distress c(o2-o3) | 23.749 | 3.901 | 3.210 | 06.088 | 0.000 |
| distress o2 | 27.480 | 0.364 | 4.497 | 75.421 | 0.000 |
| **variances** | | | | | |
| self-esteem c(o2-o3) | 047.444 | 05.610 | 0.788 | 08.457 | 0.000 |
| self-esteem o2 | 154.221 | 11.850 | 1.000 | 13.014 | 0.000 |
| distress c(o2-o3) | 032.635 | 02.957 | 0.596 | 11.035 | 0.000 |
| distress o2 | 037.348 | 03.158 | 1.000 | 11.827 | 0.000 |

*Note.* SE = standard error. O = occasion.

## APPENDIX F.7: CHECKING METRIC INVARIANCE

Please note, the below confirmatory factor models do not resemble the much more constrained sum-scores (for the mental distress, brooding and self-esteem variables) which we have used in the Chapter. In the Chapter we opted to use sum-scores, as our sample size limited the possible complexity of our models. However, sum-scores imply metric invariance over measurement occasions, as all items have the same contribution (i.e. loading/ weight = 1) to the sum-score across the measurement occasions. Therefore, below we aimed to explore whether the assumption of metric invariance is approximately met. We did not amend the factorial models based on modification indices, as it was not our aim to find the best fitting model, but to find out whether metric invariance holds over the three occasions. As can be seen in Table F.15, for all three factor models (i.e. respectively one for mental distress, brooding and self-esteem) the fit indices (i.e. CFI, TLI, RMSEA and SRMR) were largely identical when we equated the factor loadings across time (i.e. metric models = M), compared to when the factor loadings could freely vary over time (i.e. configural models = C). Moreover, for all models, the Bayesian Information Criterion (BIC) favoured the metric models. For all models, except for mental distress, the Akaike Information Criterion (AIC) favoured the metric models. The chi-squared test did not differ for the two brooding models, was inconclusive for the two self-esteem models, but revealed a significant difference between the two mental distress models. We concluded that brooding was strongly metric-invariant, self-esteem was most likely metric-invariant, and the results for mental distress were potentially in favour for the configural model, but metric invariance could not be conclusively rejected (given the stable fit indices and the BIC). Thus, as metric invariance could not be rejected, it seems acceptable to compare the mental distress, brooding and self-esteem variables across the three occasions. We did not test scalar invariance, which is the item intercept/ item mean level invariance, as we did not expect that the mental distress and RF levels would be invariant, or in other words stable, in response to the stress-inducing exams.

Table F.15
*Testing metric invariance*

| | AIC | BIC | AICw | BICw | $Chi^2$ (df) | p | robust p | CFI | TLI | RMSEA | SRMR |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Perceived distress | | | | | | | | | | | |
| C | 09588.10 | 09797.79 | 01.98% | 00.00% | 0068.99(039) | - | - | 0.97 | 0.96 | 0.04 | 0.04 |
| M | 09580.30 | 09765.32 | **98.02%** | **100.00%** | 0073.19(045) | .758 | .763 | 0.98 | 0.96 | 0.04 | 0.04 |
| Mental distress | | | | | | | | | | | |
| C | 21973.21 | 22577.60 | **89.87%** | 00.00% | 1131.77(555) | - | - | 0.89 | 0.88 | 0.05 | 0.07 |
| M | 21977.58 | 22491.51 | 10.13% | **100.00%** | 1180.14(577) | .002 | .004 | 0.89 | 0.88 | 0.05 | 0.07 |
| Brooding | | | | | | | | | | | |
| C | 11501.31 | 11760.34 | 00.08% | 00.00% | 0178.12(072) | - | - | 0.95 | 0.92 | 0.06 | 0.06 |
| M | 11487.02 | 11713.15 | **99.92%** | **100.00%** | 0179.82(080) | .992 | .995 | 0.95 | 0.94 | 0.05 | 0.06 |
| Self-esteem | | | | | | | | | | | |
| C | 29132.76 | 29638.47 | 09.95% | 00.00% | 1050.14(372) | - | - | 0.92 | 0.91 | 0.06 | 0.05 |
| M | 29128.36 | 29560.06 | **90.05%** | **100.00%** | 1081.73(390) | .044 | .076 | 0.92 | 0.91 | 0.06 | 0.06 |

*Note.* C = configural model. M = Metric or weak factorial invariance model. AIC = Akaike information criterion, BIC = Bayesian information criterion. A/BICw = weight indicator for AIC/BIC (compared to the respective other model), the higher the weight the more in favour is the model. CFI = confirmatory fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, $Chi^2$ = scaled Chi-squared test, df = degrees of freedom, p = p-value.

## APPENDIX F.8: RESULTS CONDUCTED SEPARATELY FOR HIGH POSITIVE AND LOW NEGATIVE SELF-ESTEEM

Results conducted separately for high positive and low negative self-esteem (see Tables F.16 to F.20). All effects were similar for positive and negative self-esteem, except for four. First, mental distress at occasion 2 marginally predicted more increase in positive self-esteem after exams (occasion 2 to 3; p = 0.05). Second, mental distress at occasion 1 predicted more increase in negative self-esteem during exams (occasion 1 to 2; p = 0.019). Third, while low negative self-esteem at occasion 2 only marginally predicted more decrease in distress after exams (occasion 2 to 3; p = 0.05), low negative self-esteem at occasion 1 also marginally predicted more decrease in distress from before to after exams (occasion 1 to 3; p = 0.066). All other effects were rather comparable to the effects of mental distress on general self-esteem (which includes positive and negative self-esteem; see the last three Tables in Appendix F.6).

Table F.16
*High positive and low negative self-esteem levels for the three occasions*

| | Occasion 1 M(SD), N | Occasion 2 M(SD), N | Occasion 3 M(SD), N |
|---|---|---|---|
| **General self-esteem** | 47.34(12.13), 445 | 45.88(12.40), 271 | 47.54(11.25), 278 |
| **High positive** | 25.46(5.93), 447 | 24.64(6.29), 273 | 25.58(5.66), 281 |
| **Low negative** | 21.84(6.83), 449 | 21.15(6.67), 273 | 21.90(6.43), 280 |

*Note.* M = mean, SD = standard deviation, N = sample size.

Table F.17
*Latent growth model fit*

| Model | AIC | BIC | CFI | TLI | RMSEA | SRMR | Chi² (df) | BICw | AICw |
|---|---|---|---|---|---|---|---|---|---|
| **High positive self-esteem** | | | | | | | | | |
| M1* | 5803.05 | 5827.68 | 0.98 | 0.98 | 0.09 | 0.03 | 14.73 (3) | 100% | 100% |
| M2* | 5803.05 | 5827.68 | 0.98 | 0.98 | 0.09 | 0.03 | 14.73 (3) | - | - |
| M3* | 5824.60 | 5849.22 | 0.95 | 0.95 | 0.16 | 0.05 | 36.28 (3) | 000% | 000% |
| M4* | 5836.68 | 5857.20 | 0.93 | 0.95 | 0.16 | 0.08 | 50.36 (4) | 000% | 000% |
| M1* vs M4* | | Chi² difference = 15.03, df = 1, $p < 0.001$ | | | | | | | |
| **High positive self-esteem** | | | | | | | | | |
| M1** | 5802.00 | 5830.73 | 0.99 | 0.98 | 0.10 | 0.02 | 11.68 (2) | 97.92% | 99.68% |
| M2** | 5802.00 | 5830.73 | 0.99 | 0.98 | 0.10 | 0.02 | 11.68 (2) | - | - |
| M3** | 5816.99 | 5845.72 | 0.96 | 0.94 | 0.17 | 0.05 | 26.67 (2) | 00.05% | 00.06% |
| M4** | 5813.86 | 5838.49 | 0.96 | 0.96 | 0.13 | 0.05 | 25.54 (3) | 02.02% | 00.26% |
| M1** vs M4** | | Chi² difference = NP, df = NP, $p$ = NP | | | | | | | |
| **Low negative self-esteem** | | | | | | | | | |
| M1* | NC | NC | NC | NC | NC | NC | NC | - | - |
| M2* | 6053.26 | 6077.92 | 0.99 | 0.99 | 0.06 | 0.03 | 07.32 (3) | 76.10% | 93.87% |
| M3* | 6060.36 | 6085.01 | 0.98 | 0.98 | 0.09 | 0.03 | 14.42 (3) | 02.19% | 02.70% |
| M4* | 6059.88 | 6080.43 | 0.98 | 0.99 | 0.08 | 0.03 | 15.94 (4) | 21.71% | 03.43% |
| M2* vs M4* | | Chi² difference = 4.67, df = 1, $p < 0.05$ | | | | | | | |
| **Low negative self-esteem** | | | | | | | | | |
| M1** | 6054.55 | 6083.32 | 0.99 | 0.99 | 0.07 | 0.03 | 06.61 (2) | 38.75% | 78.50% |
| M2** | 6054.55 | 6083.32 | 0.99 | 0.99 | 0.07 | 0.03 | 06.61 (2) | - | - |
| M3** | 6059.56 | 6088.32 | 0.98 | 0.98 | 0.10 | 0.03 | 11.62 (2) | 03.17% | 06.43% |
| M4** | 6057.85 | 6082.51 | 0.99 | 0.99 | 0.08 | 0.04 | 11.91 (3) | 58.07% | 15.07% |
| M1** vs M4** | | Chi² difference = NP, df = NP, $p$ = NP | | | | | | | |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. AIC = Akaike information criterion, BIC = Bayesian information

criterion, CFI = confirmatory fit index, TLI = Tucker-Lewis fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, df = degrees of freedom, p = p-value. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative. NP = not possible. NC = the model did not converge.

Table F.18
*Latent growth model summary*

| Model | Slope ld. o1 | Slope ld. o2 | Slope ld. o3 | Intercept mean | Slope mean | Residual var. o1 | Residual var. o2 | Residual var. o3 | Intercept slope cv. |
|---|---|---|---|---|---|---|---|---|---|
| **High positive self-esteem** | | | | | | | | | |
| M1* | 0.81 | 0.00 | 1.00 | 24.49 | 01.15 | 7.16 | 7.16 | 7.16 | -3.762 |
| M2* | 0.81 | 0.00 | 1.00 | 24.49 | 01.15 | 7.16 | 7.16 | 7.16 | -3.762 |
| M3* | 0.00 | 0.00 | 1.00 | 25.14 | 00.53 | 7.40 | 7.40 | 7.40 | -2.646 |
| M4* | NP | 0.00 | 1.00 | 25.27 | 00.00 | 7.50 | 7.50 | 7.50 | -0.004 |
| **High positive self-esteem** | | | | | | | | | |
| M1** | 0.47 | 0.00 | 1.00 | 24.77 | 01.05 | 5.95 | 5.95 | 5.95 | -7.072 |
| M2** | 0.47 | 0.00 | 1.00 | 24.77 | 01.05 | 5.95 | 5.95 | 5.95 | -7.072 |
| M3** | 0.00 | 0.00 | 1.00 | 25.16 | 00.51 | 6.04 | 6.04 | 6.04 | -4.556 |
| M4** | 0.31 | 0.00 | 1.00 | 25.42 | 00.00 | 5.74 | 5.74 | 5.74 | -7.523 |
| **Low negative self-esteem** | | | | | | | | | |
| M1* | NP2 | 0.00 | 1.00 | 21.48 | 00.00 | 9.01 | 9.01 | 9.01 | -0.000 |
| M2* | 0.98 | 0.00 | 1.00 | 21.10 | 00.75 | 8.88 | 8.88 | 8.88 | -0.361 |
| M3* | 0.00 | 0.00 | 1.00 | 21.61 | 00.30 | 9.03 | 9.03 | 9.03 | -1.938 |
| M4* | NP3 | 0.00 | 1.00 | 21.68 | 00.00 | 9.06 | 9.06 | 9.06 | -1.438 |
| **Low negative self-esteem** | | | | | | | | | |
| M1** | 0.53 | 0.00 | 1.00 | 21.30 | 00.74 | 8.04 | 8.04 | 8.04 | -3.655 |
| M2** | 0.53 | 0.00 | 1.00 | 21.30 | 00.74 | 8.04 | 8.04 | 8.04 | -3.654 |
| M3** | 0.00 | 0.00 | 1.00 | 21.61 | 00.29 | 8.12 | 8.12 | 8.12 | -3.157 |
| M4** | 0.28 | 0.00 | 1.00 | 21.72 | 00.00 | 7.87 | 7.87 | 7.87 | -4.325 |

*Note.* M1 = the freely estimated trajectory model. M2 = the negative homeostasis trajectory model. M3 = the growth trajectory model. M4 = the no-change model. ld. = loading, o = occasion, var. = variance, cv. = covariance. *the variance for the latent slope fixed to 0, to render it non-negative. **the variance for the latent slope constrained to >0, to render it non-negative. NP = not plausible = -51.91. NP2 = -351490.69. NP3 = -0.65.

Table F.19
*High positive self-esteem: bivariate latent change score models*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression occasion 1 to 2** | | | | | |
| self-esteem o1 → distress c(o1-o2) | -0.23 | 0.07 | -0.24 | -3.43 | 0.001 |
| distress o1 → distress c(o1-o2) | -0.57 | 0.08 | -0.58 | -7.41 | 0.000 |
| distress o1 → self-esteem c(o1-o2) | -0.08 | 0.05 | -0.15 | -1.65 | 0.099 |
| self-esteem o1 → self-esteem c(o1-o2) | -0.16 | 0.04 | -0.28 | -3.66 | 0.000 |
| **(auto)regression occasion 1 to 3** | | | | | |
| self-esteem o1 → distress c(o1-o3) | -0.09 | 0.09 | -0.07 | -0.98 | 0.327 |
| distress o1 → distress c(o1-o3) | -0.74 | 0.09 | -0.62 | -8.14 | 0.000 |
| distress o1 → self-esteem c(o1-o3) | -0.01 | 0.05 | -0.01 | -0.16 | 0.875 |
| self-esteem o1 → self-esteem c(o1-o3) | -0.26 | 0.05 | -0.40 | -5.25 | 0.000 |
| **(auto)regression occasion 2 to 3** | | | | | |
| self-esteem o2 → distress c(o2-o3) | -0.16 | 0.08 | -0.14 | -2.10 | 0.035 |
| distress o2 → distress c(o2-o3) | -0.87 | 0.09 | -0.72 | -10.04 | 0.000 |
| distress o2 → self-esteem c(o2-o3) | 0.11 | 0.06 | 0.16 | 1.96 | 0.050 |
| self-esteem o2 → self-esteem c(o2-o3) | -0.25 | 0.05 | -0.39 | -5.45 | 0.000 |

*Note.* SE = standard error. O = occasion.

Table F.20
*Low negative self-esteem: bivariate latent change score models*

| | coefficient | SE | standardized coefficient | z-value | p-value |
|---|---|---|---|---|---|
| **(auto)regression occasion 1 to 2** | | | | | |
| self-esteem o1 → distress c(o1-o2) | -0.18 | 0.05 | -0.21 | -3.27 | 0.001 |
| distress o1 → distress c(o1-o2) | -0.55 | 0.08 | -0.56 | -7.16 | 0.000 |
| distress o1 → self-esteem c(o1-o2) | -0.12 | 0.05 | -0.18 | -2.34 | 0.019 |
| self-esteem o1 → self-esteem c(o1-o2) | -0.25 | 0.04 | -0.42 | -5.75 | 0.000 |
| **(auto)regression occasion 1 to 3** | | | | | |
| self-esteem o1 → distress c(o1-o3) | -0.12 | 0.07 | -0.12 | -1.84 | 0.066 |
| distress o1 → distress c(o1-o3) | -0.78 | 0.08 | -0.65 | -9.44 | 0.000 |
| distress o1 → self-esteem c(o1-o3) | -0.04 | 0.06 | -0.06 | -0.73 | 0.465 |
| self-esteem o1 → self-esteem c(o1-o3) | -0.26 | 0.04 | -0.41 | -5.80 | 0.000 |
| **(auto)regression occasion 2 to 3** | | | | | |
| self-esteem o2 → distress c(o2-o3) | -0.15 | 0.08 | -0.14 | -1.96 | 0.050 |
| distress o2 → distress c(o2-o3) | -0.86 | 0.09 | -0.71 | -10.05 | 0.000 |
| distress o2 → self-esteem c(o2-o3) | 0.02 | 0.06 | 0.02 | 0.26 | 0.796 |
| self-esteem o2 → self-esteem c(o2-o3) | -0.25 | 0.05 | -0.37 | -4.73 | 0.000 |

*Note.* SE = standard error. O = occasion.

# APPENDIX G

# Appendix Bibliography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